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 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  FEB 27   New STN AnaVist pricing effective March 1, 2006
 NEWS  4  APR 04   STN AnaVist $500 visualization usage credit offered
 NEWS  5  MAY 10   CA/CAplus enhanced with 1900-1906 U.S. patent records
 NEWS  6  MAY 11   KOREAPAT updates resume
 NEWS  7  MAY 19   Derwent World Patents Index to be reloaded and enhanced
 NEWS  8  MAY 30   IPC 8 Rolled-up Core codes added to CA/CAplus and
                   USPATFULL/USPAT2
 NEWS  9  MAY 30   The F-Term thesaurus is now available in CA/CAplus
 NEWS 10  JUN 02   The first reclassification of IPC codes now complete in
                   INPADOC
 NEWS 11  JUN 26   TULSA/TULSA2 reloaded and enhanced with new search and
                   and display fields
 NEWS 12  JUN 28   Price changes in full-text patent databases EPFULL and PCTFULL
 NEWS 13  JUl 11   CHEMSAFE reloaded and enhanced
 NEWS 14  JUl 14   FSTA enhanced with Japanese patents
 NEWS 15  JUl 19   Coverage of Research Disclosure reinstated in DWPI

 NEWS EXPRESS  JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

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 NEWS IPC8     For general information regarding STN implementation of IPC 8
 NEWS X25      X.25 communication option no longer available

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FILE 'HOME' ENTERED AT 16:30:06 ON 08 AUG 2006

=> file reg
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 3.36       3.36

FILE 'REGISTRY' ENTERED AT 16:39:40 ON 08 AUG 2006
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STRUCTURE FILE UPDATES: 7 AUG 2006 HIGHEST RN 899508-12-4
DICTIONARY FILE UPDATES: 7 AUG 2006 HIGHEST RN 899508-12-4

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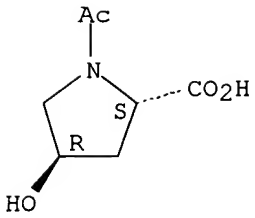
http://www.cas.org/ONLINE/UG/regprops.html

```
=> s n-acetylhydroxyproline
       6951206 N
             1 ACETYLHYDROXYPROLINE
L1           1 N-ACETYLHYDROXYPROLINE
                 (N(W)ACETYLHYDROXYPROLINE)

=> d

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   33996-33-7  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   L-Proline, 1-acetyl-4-hydroxy-, (4R)- (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   L-Proline, 1-acetyl-4-hydroxy-, trans-
CN   Proline, 1-acetyl-4-hydroxy-, L- (7CI, 8CI)
OTHER NAMES:
CN   (R)-N-Acetyl-4-hydroxy-L-proline
CN   AHP 200
CN   CO 61
CN   Jonctum
CN   N-Acetyl-4-hydroxy-L-proline
CN   N-Acetyl-4-hydroxyproline
CN   N-Acetyl-L-hydroxyproline
CN   N-Acetyl-trans-4-hydroxy-L-proline
CN   N-Acetylhydroxyproline
CN   Oxaceprol
CN   trans-N-Acetyl-4-hydroxy-L-proline
FS   STEREOSEARCH
MF   C7 H11 N O4
CI   COM
LC   STN Files:   BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS,
       CHEMINFORMRX, CHEMLIST, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT,
       IFIUDB, MEDLINE, MRCK*, PS, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL
         (*File contains numerically searchable property data)
     Other Sources:   EINECS**, NDSL**, TSCA**, WHO
         (**Enter CHEMLIST File for up-to-date regulatory information)
```

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

147 REFERENCES IN FILE CA (1907 TO DATE)
7 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
147 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> file caplus medline biosis embase

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	11.86	15.22

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FILE 'BIOSIS' ENTERED AT 16:40:06 ON 08 AUG 2006
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FILE 'EMBASE' ENTERED AT 16:40:06 ON 08 AUG 2006
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=> s l1 or oxaceprol or acetyl (l) hydroxyproline
L2 561 L1 OR OXACEPROL OR ACETYL (L) HYDROXYPROLINE

=> s l2 and (bed sore or ulcer or decubitis or decubitus)
L3 18 L2 AND (BED SORE OR ULCER OR DECUBITIS OR DECUBITUS)

=> s decubitus sore or pressure sore or pressure or bed sore or bedsore or ischial tuberosity ulcer or bed ridden or bedridden or bed rest injury or bedrest injury or air-filled bed)
UNMATCHED RIGHT PARENTHESIS 'BED)'
The number of right parentheses in a query must be equal to the number of left parentheses.

=> s decubitus sore or pressure sore or pressure or bed sore or bedsore or ischial tuberosity ulcer or bed ridden or bedridden or bed rest injury or bedrest injury or air-filled bed
L4 2787789 DECUBITUS SORE OR PRESSURE SORE OR PRESSURE OR BED SORE OR BEDSORE OR ISCHIAL TUBEROSITY ULCER OR BED RIDDEN OR BEDRIDDEN OR BED REST INJURY OR BEDREST INJURY OR AIR-FILLED BED

=> s l2 and l4
L5 23 L2 AND L4

=> dup rem l3 or l5

'L21' IS NOT VALID HERE

=> dup rem l3
PROCESSING COMPLETED FOR L3
L6 16 DUP REM L3 (2 DUPLICATES REMOVED)

=> dup rem l5
PROCESSING COMPLETED FOR L5
L7 14 DUP REM L5 (9 DUPLICATES REMOVED)

=> s l6 or l7
L8 26 L6 OR L7

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9 26 DUP REM L8 (0 DUPLICATES REMOVED)

=> focus
PROCESSING COMPLETED FOR L9
L10 26 FOCUS L9 1-

=> d ibib abs it hitstr 1-26

L10 ANSWER 1 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1974:52378 CAPLUS
DOCUMENT NUMBER: 80:52378
TITLE: Preparation of N-acetyl-L-hydroxyproline monohydrate
INVENTOR(S): Ryono, Hirokazu; Nishi, Kojiro
PATENT ASSIGNEE(S): Ajinomoto Co., Inc.
SOURCE: Jpn. Kokai Tokkyo Koho, 4 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

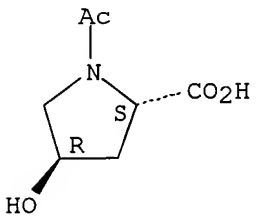
PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
JP 48085718	A2	19731113	JP 1972-12369		19720203
PRIORITY APPLN. INFO.:			JP 1972-12369	A	19720203

AB N-Acetyl-L-hydroxyproline-H2O is prepared by 1st acetylating hydroxyproline with anhydrous AcOH, and then removing most of the AcOH, and finally by adding water to the product. The antirheumatic agent N-acetyl-L-hydroxyproline is readily obtained from the stable hydrated form. Thus, 1684 g L-hydroxyproline was added to 640 ml AcOH and heated at 50-70° with constant stirring. To this mixture was added 132 g anhydrous AcOH and the mixture stirred for 45 min. AcOH was then distilled out under reduced pressure to obtain 320 ml condensed solution Half of this was combined with 320 ml H2O, and AcOH was removed as AcOH-H2O mixture by distillation to obtain a 160 ml condensate in which crystals of N-acetyl-L-hydroxyproline·H2O were formed at 0°.

IT Rheumatism
(acetylhydroxyproline for treatment of)
IT 51-35-4
RL: RCT (Reactant); RACT (Reactant or reagent)
(acetylation of)
IT 33996-33-7
RL: BIOL (Biological study)
(rheumatism treatment with)
IT 33996-33-7

RL: BIOL (Biological study)
(rheumatism treatment with)
RN 33996-33-7 CAPLUS
CN L-Proline, 1-acetyl-4-hydroxy-, (4R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L10 ANSWER 2 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:291948 CAPLUS
DOCUMENT NUMBER: 140:292668
TITLE: N-acylated hydroxyproline as preventive or remedy for bedsore
INVENTOR(S): Takeda, Toshiaki; Shimada, Kenjiro; Kawabe, Hideo; Shibasaki, Takeshi; Takahashi, Tomoya
PATENT ASSIGNEE(S): Kyowa Hakko Kogyo Co., Ltd., Japan
SOURCE: PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004028531	A1	20040408	WO 2003-JP12525	20030930
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003299074	A1	20040419	AU 2003-299074	20030930
EP 1547594	A1	20050629	EP 2003-756607	20030930
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1684679	A	20051019	CN 2003-823426	20030930
US 2006035957	A1	20060216	US 2005-529721	20050330
PRIORITY APPLN. INFO.:			JP 2002-285875 A	20020930
			WO 2003-JP12525 W	20030930

AB It is possible to provide a preventive or a remedy for bedsore which contains an N-acylated derivative of hydroxyproline or its salt. The preventive or remedy contains from 0.1 to 15 % by weight of the C1-24 acylated derivative of hydroxyproline or its salt based on the total weight For example, an ointment contained N-acetylhydroxyproline 2.5, 1,3-butylene glycol 7, triethanolamine 2.7, methylparaben 0.15, stearic acid 5, cetanol 2, cetyl palmitate 2, trimethylolpropane trioctanoate 10, lanolin 3, glycerin monostearate 0.5, paraffin oil 5, polyoxyethylene sorbitan

monostearate 3, and distilled water balance to 100 %.

IT Beverages
(N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Drug delivery systems
(capsules, soft; N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Drug delivery systems
(capsules; N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Bakery products
(cookies; N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Skin, disease
(decubitus ulcer; N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Ulcer
(decubitus; N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Drug delivery systems
(ointments, creams; N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Drug delivery systems
(ointments; N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Drug delivery systems
(powders; N-acylated hydroxyproline for prevention and treatment of bedsore)

IT Drug delivery systems
(tablets; N-acylated hydroxyproline for prevention and treatment of bedsore)

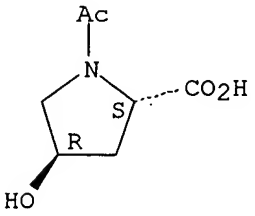
IT 33996-33-7, N-Acetylhydroxyproline
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(N-acylated hydroxyproline for prevention and treatment of bedsore)

IT 33996-33-7, N-Acetylhydroxyproline
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(N-acylated hydroxyproline for prevention and treatment of bedsore)

RN 33996-33-7 CAPLUS

CN L-Proline, 1-acetyl-4-hydroxy-, (4R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1998:247002 CAPLUS

DOCUMENT NUMBER: 128:265923

TITLE: Effects of oxaceprol on the microcirculation

in ischemia/reperfusion injury

AUTHOR(S): Harris, Anthony G.; Schropp, A.; Messmer, K.
CORPORATE SOURCE: Inst. Surgical Research, Klinikum Grosshadern, Munich, D-81366, Germany

SOURCE: European Journal of Medical Research (1998), 3(4), 182-188
CODEN: EJMRFL; ISSN: 0949-2321
PUBLISHER: I. Holzapfel Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects were examined of oxaceprol on the microcirculation of striated skin muscle. Ischemia/reperfusion injury was induced in the dorsal skin-fold chamber of the awake Syrian golden hamster by applying a 4-h complete pressure ischemia. Prior to ischemia, after 30 min, 2 h, and 24 h of reperfusion macromol. leakage, leukocyte rolling fraction, adherent leukocytes, and functional capillary d. (FCD) were assessed in a blinded study. Rhodamine 6G to stain leukocytes in vivo and FITC dextran (mol. weight 150,000) was used as a blood plasma marker. Fifteen min prior to reperfusion the animals received either an i.v. bolus infusion of oxaceprol (50 mg/kg) or an equivalent volume of saline, which was followed by a 45-min continuous infusion at the same dose. At the conclusion of the experiment samples were collected from the chamber tissue for histol. quantification of leukocyte extravasation using an esterase stain. Oxaceprol treatment resulted in a decrease of postischemic leukocyte adherence after 0.5 and 2 h of reperfusion. The histol. sections revealed a reduction in the number of extravasated leukocytes. There was a reduction of macromol. leakage and treatment also resulted in a preservation of tissue perfusion, indicated by an increase in FCD in the treatment group compared to the ischemia group. Oxaceprol protected the tissue from ischemia/reperfusion injury.

IT Adhesion, biological
Leukocyte
(decrease of postischemic leukocyte adherence after oxaceprol)

IT Circulation
(microcirculation; oxaceprol on microcirculation in ischemia/reperfusion injury)

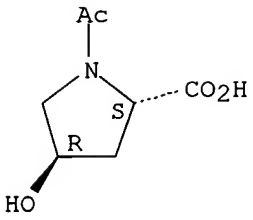
IT Antiarthritics
(oxaceprol on microcirculation in ischemia/reperfusion injury)

IT 33996-33-7, Oxaceprol
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oxaceprol on microcirculation in ischemia/reperfusion injury)

IT 33996-33-7, Oxaceprol
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oxaceprol on microcirculation in ischemia/reperfusion injury)

RN 33996-33-7 CAPLUS
CN L-Proline, 1-acetyl-4-hydroxy-, (4R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L10 ANSWER 4 OF 26 MEDLINE on STN
ACCESSION NUMBER: 2001433400 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11480608
TITLE: Oxaceprol, an atypical inhibitor of inflammation, reduces leukocyte adherence in mouse antigen-induced arthritis.
AUTHOR: Veihelmann A; Hofbauer A; Refior H J; Messmer K
CORPORATE SOURCE: Department of Orthopedics, Ludwig-Maximilians-University of Munich, Germany.. andyvei@lrz.uni-muenchen.de
SOURCE: Acta orthopaedica Scandinavica, (2001 Jun) Vol. 72, No. 3, pp. 293-8.
Journal code: 0370352. ISSN: 0001-6470.
PUB. COUNTRY: Norway
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 13 Aug 2001
Last Updated on STN: 13 Aug 2001
Entered Medline: 9 Aug 2001

AB Oxaceprol (N-acetyl-L-hydroxyproline), an atypical inhibitor of inflammation, is an established drug forjoint disease without serious side-effects. Recent studies have emphasized that oxaceprol has an effect on the microcirculation. Since the exact mechanism of action remains unclear, the aim of our study was to investigate the leukocyte-endothelial cell interactions in oxaceprol-treated mice with antigen-induced arthritis (AiA) using intravital microscopy. In our study, Balb/c mice were allocated to 4 groups (n 7, 8, 8, 8): 2 control groups with saline or oxaceprol and 2 groups of arthritic animals which received saline or oxaceprol (100 mg/kg twice a day intraperitoneally). The severity of arthritis was quantified by the transverse knee joint diameter. For the intravital fluorescence microscopy measurements on day 10 after inducing arthritis, the patella tendon was partily resected to visualize the intraarticular synovial tissue of the knee joint. The number of rolling and adherent leukocytes as well as RBC velocity and functional capillary density (FCD) were quantified in synovial microvessels. Furthermore, leukocyte infiltration was determined in the histological sections with an established score. No significant changes in mean arterial blood pressure or functional capillary density were found in any of the groups. However, the leukocyte rolling fraction and number of leukocytes adherent to the endothelium were increased in postcapillary venules of the synovium in arthritic animals (0.16 to 0.31, 78 cells/mm2 to 220 cells/mm2). In animals with AiA treated with oxaceprol, leukocyte adherence and swelling were significantly reduced in comparison to the arthritic animals treated with saline. Furthermore, the histological score showed less leukocyte infiltration in the oxaceprol treated arthritic animals. Thus, oxaceprol reduces leukocyte adherence in vivo and leukocyte infiltration in mouse AiA, indicating an effect on synovial microcirculation.

L10 ANSWER 5 OF 26 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 75175387 EMBASE
DOCUMENT NUMBER: 1975175387
TITLE: [Results with N acetyl hydroxyproline in management of stasis ulcer and delayed wound healing].
SPERIMENTAZIONE DELLA N ACETIL IDROSSIPROLINA NEL TRATTAMENTO DELLE ULCERE DA STASI E NEI RITARDI DE CICATRIZZAZIONE DELLE FERITE.
AUTHOR: Faldarini G.
CORPORATE SOURCE: Clin. Dermatol., Univ. Padova, Italy
SOURCE: G.ITAL.DERM.MINERVA DERM., (1974) Vol. 109, No. 10, pp. 538-547. .
CODEN: GIDRAK
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
013 Dermatology and Venereology
LANGUAGE: Italian
AB N acetyl hydroxyproline was administered to 25 patients, most of whom presented a stasis ulcer and delayed wound healing. The drug induced granulation in the majority of cases. No side effects were noted.

L10 ANSWER 6 OF 26 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 75097503 EMBASE
DOCUMENT NUMBER: 1975097503
TITLE: [Therapeutic trial with hydroxyproline in chronic leg ulcers].
ESTUDO TERAPEUTICO COM A HIDROXIPROLINA EM ULCERAS CRONICAS DE PERNA.
AUTHOR: Minelli L.; Schnitzler R.; Piraino R.
CORPORATE SOURCE: Setor de Dermatol., Cent. Ci. Saude, Univ. Estad. Londrina, Parana, Brazil
SOURCE: Revista Brasileira de Clinica e Terapeutica, (1974) Vol. 3, No. 6, pp. 193-198. .
CODEN: RBCTAP
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
013 Dermatology and Venereology
020 Gerontology and Geriatrics
019 Rehabilitation and Physical Medicine
030 Pharmacology
LANGUAGE: Portuguese
AB The authors accomplished an open observation of 22 patients with chronic leg ulcers of venous stasis and treated with N acetyl hydroxyproline 600 mg per day during a period of 8 wk; they enhanced the mean healing rates of the whole group which were derived from the percentage of healing at each 2 wk interval of the study and calculated close to 70% at the end of the 8 wk period; 50% of the studied cases showed total healing of ulcers before the completion of the 8 wk of therapy; 86.2% of the group benefited from the test drug, being classified as satisfactory results, a classification that included those cases with complete healing (50%) and those with definite improvement of cicatrization (36.2%). N acetyl hydroxyproline is a safe and efficacious medicament for the treatment of venous leg ulcers.

L10 ANSWER 7 OF 26 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 75085831 EMBASE
DOCUMENT NUMBER: 1975085831

TITLE: [Treatment of leg ulcers, due to venous stasis, with N acetyl hydroxyproline: double blind study].
TRATAMENTO DE ULCERAS DE PERNA, DEVIDAS A ESTASE VENOSA, POR MEIO DA N ACETIL HIDROXIPROLINA: AVALIACAO DUPLO CEGA.
AUTHOR: Zoppe A.
CORPORATE SOURCE: Dept. Cir. Vasc., Esc. Paulista Med., Sao Paulo, Brazil
SOURCE: Revista Brasileira de Clinica e Terapeutica, (1974) Vol. 3, No. 5, pp. 143-148. .
CODEN: RBCTAP
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
019 Rehabilitation and Physical Medicine
013 Dermatology and Venereology
009 Surgery
LANGUAGE: Portuguese
AB This drug or placebo was given to 40 patients with leg ulcers due to venous stasis. Complete wound healing after 10 wk of treatment was observed in 18 (90%) of 20 patients receiving N acetyl hydroxyproline. Complete healing occurred after 9 wk in one patient of the placebo group. Differences in results were significant. Laboratory investigations showed normal results. No important side effects were noted. (14 references).

L10 ANSWER 8 OF 26 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 94094817 EMBASE
DOCUMENT NUMBER: 1994094817
TITLE: [Decubitus ulcers: Medicosurgical treatment (II)].
TRATAMIENTO MEDICO-QUIRURGICO DE LAS ULCERAS POR DECUBITO (II).
AUTHOR: Marin Bertolin S.; Gonzalez Martinez R.; Garay Burdeos M.; Neira Gimenez C.; Marquina Vila P.; Amorrortu Velayos J.
CORPORATE SOURCE: Unidad de Cirugia Plastica, y Reparadora, Hospital General Universitario, Avda. Tres Cruces, s/n,46014 Valencia, Spain
SOURCE: Geriatrika, (1994) Vol. 10, No. 1, pp. 15-21. .
ISSN: 0212-9744 CODEN: GERIE5
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 013 Dermatology and Venereology
020 Gerontology and Geriatrics
037 Drug Literature Index
LANGUAGE: Spanish
SUMMARY LANGUAGE: English; Spanish
ENTRY DATE: Entered STN: 27 Apr 1994
Last Updated on STN: 27 Apr 1994
AB Pressure sores are particulary common in the elderly. Both medical and surgical treatments are possible, depending on a number of factors. The authors present their experience with different approaches.

L10 ANSWER 9 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:902714 CAPLUS
DOCUMENT NUMBER: 143:235463
TITLE: Combination of proton pump inhibitor, buffering agent, and nonsteroidal anti-inflammatory agent
INVENTOR(S): Proehl, Gerald T.; Olmstead, Kay; Hall, Warren
PATENT ASSIGNEE(S): Santarus, Inc., USA
SOURCE: PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005076987	A2	20050825	WO 2005-US3791	20050204
WO 2005076987	A3	20060608		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2005249806	A1	20051110	US 2005-51260	20050204
PRIORITY APPLN. INFO.:			US 2004-543636P	P 20040210

AB Pharmaceutical compns. comprising a proton pump inhibitor, one or more buffering agent and a nonsteroidal anti-inflammatory drug are described. Methods are described for treating gastric acid-related disorders and treating inflammatory disorders, using pharmaceutical compns. comprising a proton pump inhibitor, a buffering agent, and a nonsteroidal anti-inflammatory drug. For example, a powder for suspension formulation contained omeprazole 20 mg, ibuprofen 400 mg, sodium bicarbonate 1895 mg, Xylitol 300 (sweetener) 2000 mg, sucrose (sweetener) 1750 mg, sucralose (sweetener) 125 mg, xanthan gum 17 mg, peach flavor 47 mg, and peppermint 26 mg.

IT Pancreas, neoplasm
(Zollinger-Ellison syndrome; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Inflammation
Pancreas, disease
(acute pancreatitis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Platelet (blood)
(adhesion; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Heart, disease
(angina pectoris; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Blood vessel, disease
Wound
(associated with use of medical devices; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Heart, disease
(attack; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Drug delivery systems
(caplets; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Drug delivery systems
(capsules; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and

inflammation)
IT Newborn
(carcinogenesis or hemorrhage in; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Ischemia
(cerebral; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Intestine, neoplasm
(colorectal, reduction of risk of; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Alzheimer's disease
Antioxidants
Asthma
Atherosclerosis
Autoimmune disease
Buffers
Burn
Cardiovascular system, disease
Coating materials
Combination chemotherapy
Dermatitis
Dyspepsia
Esophagus, disease
Hepatitis
Hypertension
Immune disease
Inflammation
Influenza
Ischemia
Organ preservation
Osteoarthritis
Platelet aggregation
Platelet aggregation
Psoriasis
Respiratory system, disease
Rheumatoid arthritis
Sexual disorders
Thrombosis
Transplant rejection
(combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Amino acids, biological studies
Glycerides, biological studies
Monoglycerides
Polyoxyalkylenes, biological studies
Prostaglandins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Drug delivery systems
(controlled-release; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Artery, disease
(coronary, restenosis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Radiation
(damage; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and

inflammation)
IT Gastric acid
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(disorders; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Blood coagulation disorders
(disseminated intravascular coagulation; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Ulcer
(duodenal; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Intestine, disease
(duodenum, ulcer; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Lung, disease
(edema, associated with acute myocardial infarction; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Drug delivery systems
(effervescent, powders; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Drug delivery systems
(enteric-coated; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Esophagus, disease
Inflammation
(esophagitis, erosive; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Heart, disease
(failure; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Embolism
(fatty; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Ulcer
(gastric; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Digestive tract, disease
(gastroesophageal reflux; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Digestive tract, disease
(gastrointestinal hypersecretory disease; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Bladder, disease
(incontinence; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Heart, disease
(infarction; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Nose, disease
(inflammation; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Intestine, disease
(inflammatory; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Reperfusion
(injury; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Autoimmune disease
(insulin-dependent diabetes mellitus; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Diabetes mellitus
(insulin-dependent; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Brain, disease
(ischemia; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Neoplasm
(metastasis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Drug delivery systems
(microcapsules; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Encapsulation
(microencapsulation; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Drug delivery systems
(microspheres; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Hemorrhage
Transformation, neoplastic
(neonate; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Anti-inflammatory agents
(nonsteroidal; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Eye, disease
Inflammation
(ophthalmitis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Ear, disease
Inflammation
(otitis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Drug delivery systems
(parenterals; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Effervescent materials
(pharmaceuticals, powders; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Inflammation
Pharynx, disease
(pharyngitis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Adhesion, biological
(platelet; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Polyphosphoric acids
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(potassium salts; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Drug delivery systems
(powders, for suspensions; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Drug delivery systems
(prodrugs; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(proton pump, inhibitors; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Edema
(pulmonary, associated with acute myocardial infarction; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Infection
Inflammation
Kidney, disease
(pyelonephritis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Digestive tract, disease
(pyrosis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Esophagus, neoplasm
Lung, neoplasm
Mammary gland, neoplasm
Prostate gland, neoplasm
(reduction of risk of; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Injury
(reperfusion; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Artery, disease
(restenosis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)
IT Amino acids, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(salts, alkali metal salts; combination of proton pump inhibitor,

buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Mental and behavioral disorders
(senile psychosis; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Cell proliferation
(smooth muscle; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Muscle
(smooth, proliferation; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Brain, disease
(stroke; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Drug delivery systems
(suspensions, oral, powders for; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Drug delivery systems
(sustained-release; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Drug delivery systems
(tablets, chewable; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Drug delivery systems
(tablets; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Injury
(trauma; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT Stomach, disease
(ulcer; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT 41340-25-4, Lodine
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Etodolac; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT 24938-16-7, Eudragit EPO
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Eudragit E100; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT 9004-65-3, Methocel E5
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Methocel K4M, Sepifilm LP; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT 42924-53-8, Relafen
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nabumetone; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT 162011-90-7, Vioxx

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Rofecoxib; combination of proton pump inhibitor, buffering agent, and NSAID agent for treatment of gastric acid-related disorders and inflammation)

IT 50-33-9, Phenylbutazone, biological studies 50-78-2, Aspirin 53-86-1, Indomethacin 53-89-4, Benzpiperylon 56-87-1, L-Lysine, biological studies 57-08-9, ε-Acetamidocaproic acid 57-50-1, Sucrose, biological studies 61-68-7, Mefenamic acid 62-54-4, Calcium acetate 68-04-2, Sodium citrate 69-46-5, Calcium acetylsalicylate 72-17-3, Sodium lactate 74-79-3, L-Arginine, biological studies 77-86-1, Trometamol 79-10-7D, Acrylic acid, polymers 87-28-5, Glycol salicylate 87-99-0, Xylitol 89-45-2, Salicylsulfuric acid 89-57-6, Mesalamine 103-90-2, Paracetamol 118-55-8, Phenyl salicylate 118-57-0, Acetaminosalol 127-08-2, Potassium acetate 127-09-3, Sodium acetate 128-37-0, BHT, biological studies 129-20-4, Oxyphenbutazone 134-55-4, Phenyl acetylsalicylate 140-99-8, Calcium succinate 142-72-3, Magnesium acetate 144-55-8, Sodium bicarbonate, biological studies 147-90-0, Morpholine salicylate 150-90-3, Disodium succinate 298-14-6, Potassium bicarbonate 299-28-5, Calcium gluconate 463-79-6D, Carbonic acid, alkaline earth or Group IA metal salts 471-34-1, Calcium carbonate, biological studies 479-92-5 487-48-9, Salacetamide 489-84-9, Guaiazulene 490-79-9, Gentisic acid 497-19-8, Sodium carbonate, biological studies 515-69-5, α-Bisabolol 527-07-1, Sodium gluconate 530-78-9, Flufenamic acid 533-96-0, Sodium sesquicarbonate 546-93-0, Magnesium carbonate 549-14-4, Magnesium phthalate 550-97-0, 1-Naphthyl salicylate 552-94-3, Salsalate 556-32-1, Magnesium succinate 557-04-0, Magnesium stearate 584-08-7, Potassium carbonate 589-44-6, 3-Amino-4-hydroxybutyric acid 599-79-1, Sulfasalazine 642-72-8, Benzydamine 644-62-2, Meclofenamic acid 814-80-2, Calcium lactate 841-73-6, Bucolome 959-10-4, Xenbucin 1303-96-4, Borax ($B_4Na_2O_7.10H_2O$) 1305-62-0, Calcium hydroxide, biological studies 1309-42-8, Magnesium hydroxide 1309-48-4, Magnesium oxide, biological studies 1310-73-2, Sodium hydroxide, biological studies 1338-39-2, Span 20 1343-88-0, Magnesium silicate 1553-60-2, Ibufenac 1729-61-9, Paranyline 2055-44-9, Perisoxal 2090-64-4, Magnesium bicarbonate 2210-63-1, Mofebutazone 2316-64-5, Bromosaligenin 2438-72-4, Bufexamac 3164-34-9, Calcium tartrate 3583-64-0, Bumadizon 3615-24-5, Ramifenazone 3632-91-5, Magnesium gluconate 3983-19-5, Calcium bicarbonate 4394-00-7, Niflumic acid 5003-48-5, Benorylate 5104-49-4, Flurbiprofen 5728-52-9, Felbinac 5793-85-1, Calcium phthalate 6536-18-1, Morazone 7320-34-5, Tetrapotassium pyrophosphate 7558-79-4, Dibasic sodium phosphate 7601-54-9, Trisodium phosphate 7632-05-5, Sodium phosphate 7693-13-2, Calcium citrate 7722-88-5, Sodium pyrophosphate 7758-11-4, Dipotassium hydrogen phosphate 7758-29-4, Sodium tripolyphosphate 7778-49-6, Potassium citrate 7778-53-2, Tripotassium phosphate 7779-25-1, Magnesium citrate 7790-53-6, Potassium metaphosphate 9000-11-7, Carboxymethyl cellulose 9000-11-7D, Carboxymethyl cellulose, salts 9002-89-5, Polyvinyl alcohol 9004-38-0, Cellulose acetate phthalate 9004-57-3, Ethocel 9004-62-0, Hydroxyethyl cellulose 9004-64-2, Klucel EXF 9004-67-5, Methocel A15LV 10043-83-1, Magnesium phosphate 10103-46-5, Calcium phosphate 10197-71-4, Sodium phthalate 11121-34-9, Myverol 11137-98-7, Magnesium aluminate 11138-66-2, Xanthan gum 12040-58-3, Calcium borate 12304-65-3, Hydrotalcite 12619-64-6, Magnesium borate 12619-70-4, Cyclodextrin 12712-38-8, Potassium borate 13539-59-8, Apazone 13682-92-3 13710-19-5, Tolfenamic acid 13799-03-6, Protizinic acid 13993-65-2, Metiazinic acid 14047-56-4 14475-11-7, Sodium tartrate 15307-79-6, Voltaren 15307-86-5, Diclofenac 15551-62-9 15687-27-1, Ibuprofen 15722-48-2, Olsalazine 16068-46-5, Potassium phosphate 17465-86-0, Cavamax W8 17969-20-9, Fenclozic acid 18046-21-4, Fentiazac 18471-20-0, Ditazole 18694-40-1, Epirizole 18917-93-6, Magnesium lactate 20168-99-4, Cinmetacin 20170-20-1, Difenamizole 20187-55-7, Bendazac 20752-56-1, Magnesium tartrate 21256-18-8,

Oxaprozin 21645-51-2, Aluminum hydroxide, biological studies
22071-15-4, Ketoprofen 22131-79-9, Alclofenac 22204-53-1, Naproxen
22445-04-1 22494-42-4, Diflunisal 22760-18-5, Proquazone 23049-93-6,
Enfenamic acid 23779-99-9, Floctafenine 24237-54-5, Tinoridine
24622-72-8, Amixetrine 25322-68-3, Polyethylene glycol 25322-68-3D,
Polyethylene glycol, copolymers 25395-22-6, Salicylamide o-acetic acid
26159-34-2, Naproxen sodium 26171-23-3, Tolmetin 27035-30-9,
Oxametacin 27203-92-5, Tramadol 27214-00-2, Calcium glycerophosphate
27315-91-9, Pipebuzone 27470-51-5, Suxibuzone 29098-15-5, Terofenamate
29679-58-1, Fenoprofen 29801-94-3, Potassium phthalate 29908-03-0
30544-47-9, Etofenamate 30653-83-9, Parsalmide 30748-29-9, Feprazone
31793-07-4, Pirprofen 31842-01-0, Indoprofen 32527-55-2, Tiaramide
32808-51-8, Bucloxic acid 33005-95-7, Tiaprofenic acid 33369-31-2,
Zomepirac 33996-33-7, Oxaceprol 34148-01-1, Clidanac
34552-84-6, Isoxicam 36322-90-4, Piroxicam 36330-85-5, Fenbufen
36364-49-5, Imidazole salicylate 36981-91-6, Fepradinol 37933-78-1,
Lysine acetylsalicylate 38194-50-2, Sulindac 38957-41-4, Emorfazone
39366-43-3, Aluminum magnesium hydroxide 40828-46-4, Suprofen
40968-90-9, Potassium tartrate 42779-82-8, Clopirac 50270-33-2,
Isofezolac 51234-28-7, Benoxaprofen 51484-40-3, Difenpiramide
51579-82-9, Amfenac 51803-78-2, Nimesulide 52443-21-7, Glucametacin
52549-17-4, Pranoprofen 53164-05-9, Acemetacin 53597-27-6, Fendosal
53648-05-8, Ibuproxam 53716-49-7, Carprofen 53808-88-1, Lonazolac
54749-86-9, Thiazolinobutazone 55453-87-7, Isoxepac 55837-18-8,
Butibufen 56038-13-2, Sucralose 56187-89-4, Ximoprofen 57021-61-1,
Isonixin 57132-53-3, Proglumetacin 59804-37-4, Tenoxicam 60576-13-8,
Piketoprofen 62992-61-4, Etersalate 64425-90-7, Choline magnesium
trisalicylate 65189-78-8, Tropesin 65847-85-0, Morniflumate
66898-62-2, Talniflumate 66934-18-7, Flunoxaprofen 68767-14-6,
Loxoprofen 70374-27-5, Lomoxicam 71002-09-0, Pirazolac 71125-38-7,
Meloxicam 73590-58-6, Omeprazole 74103-06-3, Ketorolac 74711-43-6,
Zaltoprofen 74811-65-7, Ac-Di-Sol 78499-27-1, Bermoprofen
78967-07-4, Mofezolac 82821-47-4, Aminoprofen 87344-06-7, Amtolmetin
guacil 89796-99-6, Aceclofenac 90101-16-9, Droxicam 91714-94-2,
Bromfenac 92340-57-3, Hydroxyomeprazole 99464-64-9, Ampiroxicam
102625-70-7, Pantoprazole 103577-45-3, Lansoprazole 104340-86-5,
Leminoprazole 111406-87-2, Zileuton 113712-98-4, Tenatoprazole
117976-89-3, Rabeprazole
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination of proton pump inhibitor, buffering agent, and NSAID agent
for treatment of gastric acid-related disorders and inflammation)

IT 117976-90-6, Pariprazole 119141-88-7, Esomeprazole 120210-48-2,
Tenidap 161973-10-0, Perprazole 169590-42-5, Celecoxib 181695-72-7,
Valdecoxib 350507-35-6, Dontoprazole 371162-28-6, Opadry AMB
497233-50-8, Eudragit RD 100 561322-29-0, Opadry YS-1-7003
596795-01-6, Kollicoat IR 832103-67-0, Ransoprazole
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination of proton pump inhibitor, buffering agent, and NSAID agent
for treatment of gastric acid-related disorders and inflammation)

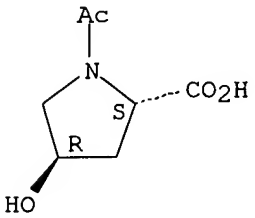
IT 329900-75-6, Cyclooxygenase II
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; combination of proton pump inhibitor, buffering agent, and
NSAID agent for treatment of gastric acid-related disorders and
inflammation)

IT 9005-25-8, Starch, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(modified; combination of proton pump inhibitor, buffering agent, and
NSAID agent for treatment of gastric acid-related disorders and
inflammation)

IT 33996-33-7, Oxaceprol
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination of proton pump inhibitor, buffering agent, and NSAID agent
for treatment of gastric acid-related disorders and inflammation)

RN 33996-33-7 CAPLUS
CN L-Proline, 1-acetyl-4-hydroxy-, (4R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



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AB Introduction. Leg ulcers are a common disease in dermatology. Their social and economic implications are important, especially when contact dermatitis complicates these wounds. Patients and methods. In this study a total of 359 patients had contact dermatitis investigations over a period of 5 years. Patch-tests were performed on 116 men and 243 women hospitalized with venous and/or arterial leg ulcers, with or without clinical appearance of periulcerous contact dermatitis. Standard patch-tests of the ICDRG (International Contact Dermatitis Research Group) were systematically performed as well as a specific series of 40 tests. Results and discussion. Positive patch tests were observed in 82.5 p. 100 of the patients, indicating a very high rate of contact allergy in patients with leg ulcers. Peru balsam, lanolin and neomycin were the most frequent culprits of positive patch-tests in this population. Five percent of patch-tests with eosin were positive in our study. This is not common in the literature. Moreover, we highlight the high rate of contact sensitization to corticosteroids in this population. According to these results and to the literature, a new series of patch-tests for leg ulcers is suggested. Conclusion. Polysensitization in patients with chronic wounds is very frequent. A series of patch-tests in leg ulcers may lead to some interesting conclusions but a good questioning of the patient is always necessary to complete this series.

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ACCESSION NUMBER: 94280194 EMBASE
DOCUMENT NUMBER: 1994280194
TITLE: [The medical-surgical treatment of the bet sore (II)]. TRATAMIENTO MEDICO-QUIRURGICO DE LAS ULCERAS POR DECUBITO (II).
AUTHOR: Martin Bertolin S.; Gonzalez Martinez R.; Garay Burdeos M.; Neira Gimenez C.; Marquina Vila P.; Amorrortu Velayos J.
CORPORATE SOURCE: Unid. de Cirugia Plastica/Reparadora, Hospital General Universitario, Avda. Tres Cruces, s/n,46014 Valencia, Spain
SOURCE: Ciencia Pharmaceutica, (1994) Vol. 4, No. 3, pp. 137-143. .
ISSN: 1131-5253 CODEN: CIPHEA
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 013 Dermatology and Venereology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: Spanish
SUMMARY LANGUAGE: English; Spanish
ENTRY DATE: Entered STN: 6 Oct 1994
Last Updated on STN: 6 Oct 1994

AB Pressure sores are particularly common in the elderly. Both medical and surgical treatments are possible, depending on a number of factors. The authors present their experience with different approaches.

L10 ANSWER 12 OF 26 MEDLINE on STN

ACCESSION NUMBER: 81131896 MEDLINE
DOCUMENT NUMBER: PubMed ID: 555265
TITLE: [Action of N-acetyl-hydroxyproline in the treatment of cutaneous ulcerative lesions]. L'azione della N-acetil-idrossiprolina nella guarigione delle lesioni ulcerative cutanee.
AUTHOR: Famulari C; Monaco M; Versaci A; Perri S; Terranova M L; Cuzzocrea D
SOURCE: Annali italiani di chirurgia, (1979) Vol. 51, No. 5, pp. 527-36.
Journal code: 0372343. ISSN: 0003-469X.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198104
ENTRY DATE: Entered STN: 16 Mar 1990
Last Updated on STN: 16 Mar 1990
Entered Medline: 13 Apr 1981

L10 ANSWER 13 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1982:400749 CAPLUS
DOCUMENT NUMBER: 97:749
TITLE: Angiotensin-converting enzyme inhibitors as antihypertensives
PATENT ASSIGNEE(S): University of Miami, USA
SOURCE: Jpn. Kokai Tokkyo Koho, 14 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 8
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

JP 56164115	A2	19811217	JP 1981-30429	19810303
JP 03000386	B4	19910107		
US 4692459	A	19870908	US 1980-121188	19800303
US 4692437	A	19870908	US 1980-156749	19800605
HU 35246	A2	19850628	HU 1981-529	19810303
HU 199786	B	19900328		
ES 501730	A2	19820401	ES 1981-501730	19810428
AT 8102051	A	19891215	AT 1981-2051	19810508
AT 390796	B	19900625		
DD 159426	C	19830309	DD 1981-230574	19810604

PRIORITY APPLN. INFO.:

US 1980-121188	A	19800303
US 1980-156749	A	19800605
US 1978-941289	A1	19780911
US 1978-958180	A1	19781106
US 1979-64897	A2	19790814
US 1979-64898	A2	19790814
US 1979-64899	A2	19790814
US 1979-64900	A2	19790814
US 1979-64901	A2	19790814
US 1979-64902	A2	19790814
US 1979-64903	A2	19790814
US 1980-116950	A2	19800130
US 1980-116951	A2	19800130
US 1980-128953	A	19800310
US 1980-158278	A	19800610

AB RAS(CH2)nCHR1C(O)R2 (R = H, formyl, acetyl, etc.; A = L-phenylalanyl, D-alanyl, D-tyrosyl, etc.; R1 = H or Me; R2 = L-proline, L-3-hydroxyproline, etc.; n = 0 or 1) are inhibitors of angiotensin I-converting enzyme [9015-82-1] and are antihypertensives. Thus, Nα-[3-Nα-benzoyl-DL-phenylalanylthio)-2-D-methylpropanoyl]-L-proline Na salt [81969-22-4] (10 μmol/kg, orally) given to rats pretreated with angiotensin I decreased blood pressure by 76% in 15 min.

IT Antihypertensives
(peptide angiotensin-converting enzyme inhibitors as)

IT 920-46-7
RL: RCT (Reactant); RACT (Reactant or reagent)
(acylation by, of glutamic acid)

IT 56-86-0, reactions
RL: RCT (Reactant); RACT (Reactant or reagent)
(acylation of, with methacryloyl chloride)

IT 13734-34-4 18942-49-9
RL: RCT (Reactant); RACT (Reactant or reagent)
(esterification of, with mercaptopropanoylproline derivative)

IT 63250-36-2
RL: RCT (Reactant); RACT (Reactant or reagent)
(esterification of, with phenylalanine derivative)

IT 9015-82-1
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors, as antihypertensives)

IT 80125-04-8P 80125-06-0P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and benzoylation of)

IT 75691-90-6P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and deblocking of)

IT 80079-49-8P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and reaction with thioacetic acid)

IT 78039-42-6P 81904-39-4P 81904-40-7P 81904-41-8P 81924-39-2P
81968-32-3P 81968-33-4P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)
IT 75692-02-3P 80079-50-1P 80125-07-1P 81968-28-7P 81968-29-8P
81968-30-1P 81969-22-4P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of, as angiotensin-converting enzyme inhibitor-antihypertensive)
IT 23912-64-3
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with [(acetylthio)propanoyl]proline derivative)
IT 507-09-5, reactions
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with methacryloyloxoproline)

L10 ANSWER 14 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1981:189899 CAPLUS
DOCUMENT NUMBER: 94:189899
TITLE: Enzymuria (the output of gamma-glutamyl-transpeptidase and of N-acetyl-beta-D-glucosaminidase) in the course of experimental renovascular hypertension
AUTHOR(S): Malyusz, M.; Braun, D.
CORPORATE SOURCE: Physiol. Inst., Univ. Kiel, Kiel, D-2300, Fed. Rep. Ger.
SOURCE: Enzyme (1981), 26(1), 32-42
CODEN: ENZYBT; ISSN: 0013-9432
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The urinary output of γ-glutamyl-transpeptidase (γGT) and of N-acetyl-β-D-glucosaminidase (NAG) was studied in rats with 2-kidney Goldblatt hypertension 3-6, 16-19, and 30-33 wk after eliciting high blood pressure. The γGT excretion rate of normotensive males was higher than that of females, while the activity of the renal tissue was on the same level. The γGT output of hypertensive males was elevated in the early and in the middle stages of the disease, it was subnormal in the late stage. In females, γGT output increased only in animals with excessively high blood pressure (>200 mmHg). The γGT output correlated with the tissue activity in males only. In all animals, there was an inverse, linear correlation between tissue γGT activity and the hydroxyproline content. The pattern of the NAG output was similar to that of γGT, however, excretion of NAG showed no sex differences and remained high in the late stage of the disease, too. Nephrosclerosis was less pronounced in female Goldblatt rats than in males.
IT Urine
(glutamyltranspeptidase and acetylglucosaminidase of, in renal hypertension, sex in relation to)
IT Sex
(glutamyltranspeptidase of urine in renal hypertension in relation to)
IT Kidney, composition
(hydroxyproline of, γ-glutamyltranspeptidase of urine in renal hypertension in relation to)
IT Hypertension
(renal, glutamyltranspeptidase and acetylglucosaminidase of urine in, sex in relation to)
IT 51-35-4
RL: BIOL (Biological study)
(of kidney, glutamyltranspeptidase of urine in renal hypertension in relation to)
IT 9012-33-3
RL: BIOL (Biological study)
(of urine, in renal hypertension)

IT 9046-27-9
RL: BIOL (Biological study)
(of urine, in renal hypertension, kidney hydroxyproline and sex in relation to)

L10 ANSWER 15 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:535126 CAPLUS
DOCUMENT NUMBER: 133:150919
TITLE: Preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors
INVENTOR(S): Costanzo, Michael J.; Maryanoff, Bruce E.; Yabut, Stephen C.
PATENT ASSIGNEE(S): Ortho-McNeil Pharmaceutical, Inc., USA
SOURCE: PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000044733	A1	20000803	WO 2000-US883	20000113
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2361479	AA	20000803	CA 2000-2361479	20000113
EP 1147097	A1	20011024	EP 2000-909902	20000113
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
TR 200102766	T2	20011221	TR 2001-2766	20000113
BR 2000007778	A	20020604	BR 2000-7778	20000113
EE 200100391	A	20021015	EE 2001-391	20000113
JP 2002535394	T2	20021022	JP 2000-595989	20000113
US 6469036	B1	20021022	US 2000-482802	20000113
TW 229669	B1	20050321	TW 2000-89101335	20000224
NO 2001003666	A	20010926	NO 2001-3666	20010726
BG 105762	A	20020329	BG 2001-105762	20010801
HR 2001000601	A1	20020831	HR 2001-601	20010813
ZA 2001006995	A	20021125	ZA 2001-6995	20010823
US 2003008829	A1	20030109	US 2002-205355	20020725
PRIORITY APPLN. INFO.:			US 1999-117602P	P 19990127
			US 2000-482802	A3 20000113
			WO 2000-US883	W 20000113

OTHER SOURCE(S): MARPAT 133:150919
AB Peptidyl heterocyclic ketones A-NRCR1R2CO-E [A = substituted cycloalkylcarbonyl, norbornanecarbonyl, norbornenecarbonyl, adamantanecarbonyl, arylcarbonyl, heteroarylcarbonyl, aminoalkylcarbonyl, an amino acid or dipeptide residue, etc.; R, R1 = H, alkyl; R2 = amino-, guanidino-, alkylguanidino-, dialkylguanidino-, amidino-, alkylamidino-, dialkylamidino-, or alkoxyalkyl, (un)substituted Ph, benzyl, pyridyl, pyridyl-, pyrimidyl-, triazinyl-, or imidazoalkyl, imidazolinyl-, N-amidinopiperazinyl-, hydroxy-, alkylamino-, dialkylamino-, N-amidinopiperidinyl-, or 4-aminocyclohexylalkyl; E = (un)substituted heterocyclyl] and their pharmaceutically acceptable salts and prodrugs were prepared as tryptase inhibitors and are therefore effective for the prevention and treatment of inflammatory diseases associated with the respiratory tract, such as asthma and allergic rhinitis. Thus,

(2S,4R)-1-acetyl-N-[(1S)-4-[(aminoiminomethyl)amino]-1-(2-benzothiazolylcarbonyl)butyl]-4-hydroxy-2-pyrrolidinecarboxamide was prepared by a seven-step procedure starting from Boc-Arg(Ts)-OH (Boc, tert-butoxycarbonyl, Ts = tosyl), benzothiazole, and trans-1-acetyl-4-benzyloxyl-L-proline and showed IC50 = 0.036 ± 0.031 μM for inhibition of tryptase.

IT Intestine, disease
(Crohn's; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Eye, disease
(allergic conjunctivitis; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Nose
(allergic rhinitis; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Edema
(angioneurotic; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Lung, disease
(chronic obstructive; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Kidney, disease
(glomerulonephritis; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Skin, disease
(hyperproliferation; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Intestine, disease
(inflammatory; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Kidney, disease
(nephritis; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Pancreas, disease
(pancreatitis; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Ulcer
(peptic; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Anaphylaxis
Anti-inflammatory agents
Antiasthmatics
Atherosclerosis
Cirrhosis
Dermatitis
Eczema
Fibrosis
Gout
Osteoarthritis
Psoriasis
Rheumatoid arthritis
(preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Amino acids, preparation
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT Artery, disease
(restenosis; preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT 287182-55-2P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use);

BIOL (Biological study); PREP (Preparation); USES (Uses)
(G45prepn. of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT 287182-50-7P 287182-51-8P 287182-52-9P 287182-53-0P 287182-54-1P
287182-56-3P 287182-57-4P 287182-58-5P 287182-59-6P 287182-60-9P
287182-61-0P 287182-62-1P 287182-63-2P 287182-64-3P 287182-65-4P
287182-66-5P 287182-67-6P 287182-68-7P 287182-69-8P 287182-70-1P
287182-71-2P 287182-72-3P 287182-73-4P 287182-74-5P 287182-75-6P
287182-76-7P 287182-77-8P 287182-78-9P 287182-79-0P 287182-80-3P
287182-81-4P 287182-82-5P 287182-83-6P 287182-84-7P 287182-85-8P
287182-86-9P 287182-87-0P 287182-88-1P 287183-00-0P 287194-95-0P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT 9002-07-7, Trypsin 97501-93-4, Tryptase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT 68-95-1, n-Acetyl L-proline 85-46-1, 1-Naphthalenesulfonyl chloride
95-14-7, 1H-Benzotriazole 96-81-1 98-98-6, 2-Pyridinecarboxylic acid
543-24-8, n-Acetylglycine 638-32-4, Succinamic acid 1776-53-0
2577-48-2, L-Proline methyl ester 2812-46-6 5888-91-5,
n-Acetylsarcosine 6294-84-4 13836-37-8 18822-58-7, o-tert-Butyl L serine 25137-01-3, Ethyl r nipecotate 33294-81-4 41324-66-7,
L-Proline benzyl ester 51052-78-9, 4-Piperidineacetic acid 51077-01-1,
n-Tosyl L-Proline 58695-41-3 74411-98-6 82717-40-6 97290-54-5,
Cyclohexanepropanoic acid, α-(acetylamino)-, (αS)-
102185-38-6 111555-81-8 151275-35-3 174894-05-4 174960-90-8
210420-92-1 287182-93-8 287182-95-0
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

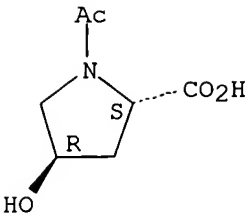
IT 33996-33-7P 61936-38-7P 119993-55-4P 140894-62-8P
142801-55-6P 179746-22-6P 182964-78-9P 182964-84-7P 186181-82-8P
186181-83-9P 201006-62-4P 203453-39-8P 287182-89-2P 287182-90-5P
287182-91-6P 287182-92-7P 287182-94-9P 287182-96-1P 287182-97-2P
287182-98-3P 287182-99-4P 287183-01-1P 287183-02-2P 287194-96-1P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT 50-02-2, Dexamethasone 50-23-7, Hydrocortisone 53-03-2, Prednisone
58-08-2, Caffeine, biological studies 58-55-9, Theophylline, biological studies 83-67-0, Theobromine 124-94-7, Triamcinolone 317-34-0,
Aminophylline 13392-18-2, Fenoterol 16110-51-3, Cromolyn 18559-94-9,
Albuterol 23031-25-6, Terbutaline 69049-73-6, Nedocromil 73573-87-2,
Formoterol
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

IT 33996-33-7P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of peptidyl heterocyclic ketones useful as tryptase inhibitors)

RN 33996-33-7 CAPLUS
CN L-Proline, 1-acetyl-4-hydroxy-, (4R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



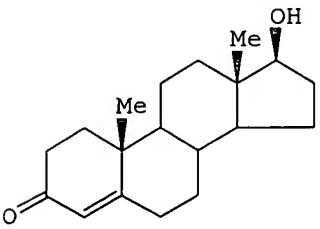
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 16 OF 26 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 77015600 EMBASE
DOCUMENT NUMBER: 1977015600
TITLE: [Jonctum (oxaceprol) in chronic ulcers of the lower extremities. Double blind study].
EL JONCTUM EN LAS ULCERAS CRONICAS DE LAS EXTREMIDADES INFERIORES. ESTUDIO DOBLE A CIEGAS.
AUTHOR: Goihman Yahr M.; Noya Leon A.; Rojas A.; Convit J.
CORPORATE SOURCE: Inst. Nac. Dermatol., Caracas, Venezuela
SOURCE: Medicina Cutanea Ibero-Latino-Americana, (1974) Vol. 2, No. 3, pp. 227-231. .
CODEN: MCILBI
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
013 Dermatology and Venereology
030 Pharmacology
LANGUAGE: Spanish
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L10 ANSWER 17 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1983:416806 CAPLUS
DOCUMENT NUMBER: 99:16806
TITLE: Effect of castration and testosterone substitution on the urinary output of gamma-glutamyl transpeptidase and of N-acetyl-beta-D-glucosaminidase of male rats with renovascular hypertension
AUTHOR(S): Malyusz, M.; Ehrens, H. J.
CORPORATE SOURCE: Physiol. Inst., Univ. Kiel, Kiel, Fed. Rep. Ger.
SOURCE: Enzyme (1983), 29(2), 93-9
CODEN: ENZYBT; ISSN: 0013-9432
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB The effect of castration of male rats with exptl. renal hypertension (2-kidney Goldblatt hypertension) was studied on the severity of the hypertension and on the urinary output of γ-glutamyl transpeptidase

(γGT) [9046-27-9] and of N- acetyl-β-D-glucosaminidase (NAG) [9012-33-3]. Castration was carried out immediately after clamping 1 renal artery. Some of the castrates received testosterone (I) [58-22-0] substitution from the 3rd postoperative week onwards. Hypertension as well as urinary enzyme were less pronounced in castrates than in uncastrated males or I-substituted rats. In all animals studied the γGT excretion rate showed a pos. correlation with the blood pressure. The output of γGT and NAG as well as the specific γGT activity of the renal membrane fraction was lower in castrates than in uncastrated males or in substituted castrates. In uncastrated males and in I-substituted castrates, the daily NAG output correlated directly with the renal hydroxyproline [51-35-4] content. No such correlation was found in castrated males. The kidneys of castrates and I-substituted castrates contained less hydroxyproline than did those of uncastrated males.

IT Urine
(acetylglucosaminidase and glutamyl transpeptidase excretion in, in hypertension, testosterone effect on)

IT Kidney, composition
(hydroxyproline and protein of, in hypertension, testosterone effect on)

IT Proteins
RL: BIOL (Biological study)
(of kidney, in hypertension, testosterone effect on)

IT Hypertension
(renal, acetylglucosaminidase and glutamyl transpeptidase excretion in urine in, testosterone effect on)

IT 58-22-0
RL: BIOL (Biological study)
(acetylglucosaminidase and glutamyl transpeptidase of urine response to, in hypertension)

IT 51-35-4
RL: BIOL (Biological study)
(of kidney, in hypertension, testosterone effect on, acetylglucosaminidase excretion in urine in relation to)

IT 9012-33-3 9046-27-9
RL: BIOL (Biological study)
(urinary excretion of, in hypertension, testosterone effect on)

L10 ANSWER 18 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1989:497769 CAPLUS
DOCUMENT NUMBER: 111:97769
TITLE: Pseudopoly(amino acids): a study of the synthesis and characterization of poly(trans-4-hydroxy-N-acyl-L-proline esters)
AUTHOR(S): Kwon, Heewon Yu; Langer, Robert
CORPORATE SOURCE: Dep. Chem. Eng., Massachusetts Inst. Technol., Cambridge, MA, 02139, USA
SOURCE: Macromolecules (1989), 22(8), 3250-5
CODEN: MAMOBX; ISSN: 0024-9297
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Polyesters of trans-4-hydroxy-N-acyl-L-proline Me esters in which the pendant acyl groups were ethanoyl, 2,2-dimethylpropanoyl, hexanoyl, decanoyl, tetradecanoyl, and hexadecanoyl were prepared and characterized. Weight-average mol. wts. >40,000 were obtained via ester interchange using 1 mol% Ti isopropoxide as catalyst at 180° for 20-24 h. Different pendant groups on the monomers profoundly affected the polymerizability and the polyester properties. When the length of the acyl group increased, the mol. weight as well as d.p. increased. A declining trend in the glass temperature of the polymers was observed with increasing acyl group chain length. Mol.

weight data obtained from gel chromatog. and vapor pressure osmometry suggested that the polymesters assumed a rodlet-like conformation in solution

IT Polymerization catalysts
(for hydroxy(acyl)proline Me esters)

IT Glass temperature and transition
(of poly(hydroxyacylproline Me esters))

IT Amino acids, esters
RL: SPN (Synthetic preparation); PREP (Preparation)
(hydroxy, Me esters, polymers, preparation and characterization of)

IT Polyesters, preparation
RL: SPN (Synthetic preparation); PREP (Preparation)
(hydroxyproline-based, preparation and characterization of)

IT 104-15-4, uses and miscellaneous 301-04-2 471-34-1, Carbonic acid calcium salt (1:1), uses and miscellaneous 543-90-8 546-68-9 555-31-7 556-91-2 557-20-0 557-34-6 865-47-4 1304-28-5, Barium oxide (BaO), uses and miscellaneous 1304-76-3, Bismuth oxide (Bi2O3), uses and miscellaneous 1305-78-8, Calcium oxide (CaO), uses and miscellaneous 1309-64-4, Antimony oxide (Sb2O3), uses and miscellaneous 7705-08-0, Iron chloride (FeCl3), uses and miscellaneous 14024-17-0 23355-24-0
RL: CAT (Catalyst use); USES (Uses)
(catalysts, for polymerization of hydroxy(oxohexadecyl)proline Me ester)

IT 120687-16-3P 120687-17-4P 120687-18-5P 120687-19-6P 120687-20-9P 120687-21-0P 120687-22-1P 120687-23-2P 120687-24-3P 120687-25-4P 120687-26-5P 190960-11-3P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation and characterization of)

IT 33996-33-7P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and esterification of)

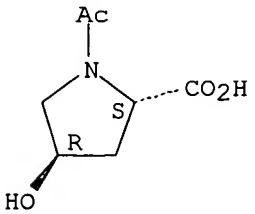
IT 67943-19-5P 99465-82-4P 106231-83-8P 120687-13-0P 120687-14-1P 120687-15-2P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and polymerization of)

IT 33996-33-7P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and esterification of)

RN 33996-33-7 CAPLUS

CN L-Proline, 1-acetyl-4-hydroxy-, (4R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L10 ANSWER 19 OF 26 MEDLINE on STN
ACCESSION NUMBER: 89253561 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3248820
TITLE: [Leg ulcers caused by prolidase deficiency].
Ulcere agli arti inferiori da deficit di prolidasi.
AUTHOR: Pasolini G; Pancera C; Manganoni A M; Cetta G; Zanaboni G

SOURCE: Giornale italiano di dermatologia e venereologia : organo ufficiale, Societa italiana di dermatologia e sifilografia, (1988 Oct) Vol. 123, No. 10, pp. 493-6.
Journal code: 8102852. ISSN: 0026-4741.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198907
ENTRY DATE: Entered STN: 6 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 10 Jul 1989

L10 ANSWER 20 OF 26 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 83061414 EMBASE
DOCUMENT NUMBER: 1983061414
TITLE: [Prolidase and manganese deficiency. A case report. Diagnosis and treatment].
DEFICIT EN PROLIDASE ET EN MANGANESE. A PROPOS D'UNE OBSERVATION: DIAGNOSTIC ET TRAITEMENT.
AUTHOR: Larregue M.; Charpentier C.; Laidet B.; et al.
CORPORATE SOURCE: Serv. Dermatol., Hotel-Dieu, F 86000 Poitiers, France
SOURCE: Annales de Dermatologie et de Venereologie, (1982) Vol. 109, No. 8, pp. 667-678. .
CODEN: ADVED7
COUNTRY: France
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
013 Dermatology and Venereology
022 Human Genetics
LANGUAGE: French
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB Prolidase deficiency, transmitted on an autosomic recessive mode upsets skin healing and facilitates the occurrence of chronic cutaneous ulcerations. A 36-year-old woman has been followed since the age of 12 for ulcerations and erythematous erysipelatoid plaques of the lower limbs. Two episodes of agranulocytosis were induced by intake of sulfonamides at the age of 17. The same incident had been observed in her aunt. As the aetiological research of ulcers was negative, a prolidase deficit was suspected. The diagnosis is ascertained by the existence of an immunopeptiduria of 5 mmol/24 hours (normally absent). The predominating dipeptides are glycylproline and phenylalanine proline. R-hydroxyproline dipeptides were present to a lesser degree. Urinary hydrolysis showed elevation of free proline (X 10) and hydroxyproline (X 6). Dosage of erythrocyte prolidase evidenced an activity 2 p. 100 of the normal in one case and 55 p. 100 and 49 p. 100 in the parents. Treatment by cofactors of prolidase (vitamin C and manganese) reduced immunopeptiduria, suppressed inflammatory outbreaks and allowed a transient cicatrisation. This tenth case of prolidase deficiency underlines the character of the disease: recurrent ulcers (7/10), erysipelatoid plaques (3/10), ecchymosis (4/10), telangiectatic scars (7/10), edema (1/10), early canitias (1/10). Partial correction by cofactors evokes a prolidase deficiency by inactivation of the enzyme activating systems.

L10 ANSWER 21 OF 26 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 94272387 EMBASE
DOCUMENT NUMBER: 1994272387
TITLE: [Means of cutaneous healing].

LES CICATRISANTS CUTANES.
AUTHOR: Mallet V.; Lemarchand-Venencie F.
CORPORATE SOURCE: Clinique des Maladies Cutanees, Service du Professeur L. Dubertret, Hopital Saint-Louis,75475 Paris Cedex 10, France
SOURCE: Revue du Praticien, (1994) Vol. 44, No. 13, pp. 1781-1785.
.
ISSN: 0035-2640 CODEN: REPRA3
COUNTRY: France
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 013 Dermatology and Venereology
037 Drug Literature Index
LANGUAGE: French
SUMMARY LANGUAGE: French; English
ENTRY DATE: Entered STN: 6 Oct 1994
Last Updated on STN: 6 Oct 1994

AB Cutaneous healing is an important field of dermatology for it concerns superficial wound, as well as little surgery action, leg ulcer, eschar or burn. In spite of the claiming of their healing properties and their profusion, only a few have been tested and have proved their efficiency. Use precautions must be complied with paying the highest attention among others to the condition of the wound before product applying, the sensitization risk and the systemic risk particularly for young child. After topics, colloids appeared about ten years ago. New technics in development are reflecting, the research perseverance in dermatology.

L10 ANSWER 22 OF 26 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1991:559744 CAPLUS
DOCUMENT NUMBER: 115:159744
TITLE: Synthesis and biological activity of O-glycosylated morphiceptin analogs
AUTHOR(S): Bardaji, Eduard; Torres, Joseph L.; Clapes, Pere; Albericio, Fernando; Barany, George; Rodriguez, Raquel E.; Sacristan, Maria P.; Valencia, Gregorio
CORPORATE SOURCE: Unit Pept. Chem. Biochem., CSIC, Barcelona, 08034, Spain
SOURCE: Journal of the Chemical Society, Perkin Transactions 1: Organic and Bio-Organic Chemistry (1972-1999) (1991), (7), 1755-9
CODEN: JCPRB4; ISSN: 0300-922X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB H-Tyr-Pro-Phe-Hyp(R)-NH2 (I; R = H, tetra-O-acetyl-β-D-galactopyranosyl, tetra-O-acetyl-β-D-glucopyranosyl) were obtained using 9-fluorenylmethoxycarbonyl solid-phase chemical and mild conditions for cleavage from a tris(alkoxy)benzylamide (PAL) resin. I were evaluated in the guinea pig ileum in vitro assay and in in vivo tail-flick and paw-pressure antinociceptive tests after intrathecal administration in rats. The substitutions resulted in an unexpected decrease in biol. activity with respect to morphiceptin.

IT Analgesics
(hydroxyproline analogs of morphiceptin)
IT 13504-85-3
RL: RCT (Reactant); RACT (Reactant or reagent)
(esterification of)
IT 604-69-3, Penta-O-acetyl-β-D-glucopyranose 4163-60-4, Penta-O-acetyl-β-D-galactopyranose
RL: RCT (Reactant); RACT (Reactant or reagent)
(glycosidation by, of hydroxyproline)
IT 74135-04-9DP, Morphiceptin, hydroxyproline analogs 125355-38-6P 125355-39-7P 136137-51-4P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological

study); PREP (Preparation)
(preparation and analgesic activity of)
IT 80134-55-0P 136233-12-0P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and deblocking of)
IT 136137-52-5P 136233-13-1P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and fluorenylmethoxycarbonylation of)
IT 13500-53-3P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and glycosidation of)
IT 125355-40-0P 125355-41-1P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation and solid-phase peptide synthesis with)

L10 ANSWER 23 OF 26 MEDLINE on STN
ACCESSION NUMBER: 87308546 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3624363
TITLE: Determination of alpha-alkyl-alpha-amino acids and alpha-amino alcohols by chiral-phase capillary gas chromatography and reverse-phase high-performance liquid chromatography.
AUTHOR: Bruckner H; Bosch I; Graser T; Furst P
SOURCE: Journal of chromatography, (1987 Jun 12) Vol. 395, pp. 569-90.
Journal code: 0427043. ISSN: 0021-9673.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198710
ENTRY DATE: Entered STN: 5 Mar 1990
Last Updated on STN: 5 Mar 1990
Entered Medline: 22 Oct 1987
AB The enantiomeric resolution by fused-silica capillary gas-liquid chromatography (GLC) of non-protein DL-alpha-alkyl-alpha-amino acids of the structure H2NCR1R2COOH (R1 = alkyl, R2 = alkyl, alkaryl) was investigated by using chiral [L-valine-tert.-butylamide, linked to a statistical polymer of dimethylsiloxane and (2-carboxypropyl)methylsiloxane, Chirasil-L-Val, and XE-60-S-Val-S-alpha-phenylethylamide] and non-chiral (methylphenylcyanopropylvinylpolysiloxane, CP-Sil-19 stationary phases. To evaluate the resolution coefficients, N-acylamino acid n-propyl esters (acyl = acetyl, propionyl, trifluoroacetyl, pentafluoropropionyl, heptafluorobutyryl) and diastereomeric esters with S(-)-2-methyl-1-butanol, S(+)-2-butanol and S(+)-2-octanol were used. Although alpha-alkyl-alpha-amino acids in general gave lower resolution coefficients than the enantiomers of protein amino acids, most alpha-alkyl-alpha-amino acids could be resolved by using suitable derivatization procedures and, preferably, isothermal conditions. In addition, a number of DL-alpha-alkyl-alpha-amino acids could be separated by ligand-exchange chromatography (L-hydroxyproline /Cu2+) by both thin-layer chromatography (Chiralplate) and high-performance liquid chromatography (HPLC) (Nucleosil Chiral-1). Further, a standard mixture composed of fifteen alphá-amino acids and eleven alpha-amino alcohols could be completely separated by C18 HPLC after derivatization with o-phthaldialdehyde-2-mercaptoethanol (OPA-2-ME). The time and temperature dependences of the relative fluorescence of the adducts were investigated kinetically.

L10 ANSWER 24 OF 26 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN
ACCESSION NUMBER: 2004:93061 BIOSIS
DOCUMENT NUMBER: PREV200400086253
TITLE: AC-SDKP attenuates renal damage in salt sensitive hypertension.
AUTHOR(S): Wang, Dahai [Reprint Author]; Yang, Xiao-Ping [Reprint Author]; Peng, Hongmei [Reprint Author]; Rhaleb, Nour-Eddine [Reprint Author]; Beierwaltes, Walliam H. [Reprint Author]; Carretero, Oscar A. [Reprint Author]
CORPORATE SOURCE: Hypertensin and Vascular Research, Henry Ford Hospital, Detroit, MI, USA
SOURCE: Journal of the American Society of Nephrology, (November 2003) Vol. 14, No. Abstracts Issue, pp. 118A. print.
Meeting Info.: Meeting of the American Society of Nephrology Renal Week. San Diego, CA, USA. November 12-17, 2003. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Feb 2004
Last Updated on STN: 11 Feb 2004
IT Major Concepts
Cardiovascular System (Transport and Circulation); Urinary System (Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
kidney: excretory system; mesangial cell: excretory system, proliferation
IT Diseases
hypertension: vascular disease
Hypertension (MeSH)
IT Diseases
renal damage: urologic disease
IT Diseases
renal fibrosis: urologic disease
IT Chemicals & Biochemicals
N-acetyl-seryl-aspartyl-lysyl-proline [Ac-SDKP]; albumin: excretion; collagen: synthesis; creatinine; hydroxyproline; sodium chloride
IT Miscellaneous Descriptors
blood pressure [BP]; glomerular filtration rate [GFR]; urinary albumin excretion rate
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rat (common): strain-Dahl salt-sensitive
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
RN 120081-14-3 (N-acetyl-seryl-aspartyl-lysyl-proline)
120081-14-3 (Ac-SDKP)
60-27-5 (creatinine)
51-35-4Q (hydroxyproline)
6912-67-0Q (hydroxyproline)
7647-14-5 (sodium chloride)

L10 ANSWER 25 OF 26 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2004150519 EMBASE
TITLE: [Clinical trials published in Medicina Cutanea Ibero-Latino-Americana between 1970 and 2000].

ENSAYOS CLINICOS PUBLICADOS EN MEDICINA CUTANEA IBERO-LATINO-AMERICANA ENTRE 1970 Y 2000.

AUTHOR: Gonzalez Castro U.
CORPORATE SOURCE: U. Gonzalez Castro, Servicio de Dermatologia, Clinica Plato, Plato, 21, 08006 Barcelona, Spain. 24998ugc@comb.es
SOURCE: Medicina Cutanea Ibero-Latino-Americana, (2002) Vol. 30, No. 6, pp. 287-292. .
Refs: 38
ISSN: 0210-5187 CODEN: MCILBI
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 013 Dermatology and Venereology
017 Public Health, Social Medicine and Epidemiology
037 Drug Literature Index
LANGUAGE: Spanish
SUMMARY LANGUAGE: English; Spanish
ENTRY DATE: Entered STN: 6 May 2004
Last Updated on STN: 6 May 2004

AB Background. The practice of evidence-based medicine requires efficient access to information from clinical trials (CT). Objectives. To locate, with the greatest possible exhaustivity, all CT published in Medicina Cutanea Ibero-Latino-Americana (MCILA) between 1970 and 2000 and to describe their characteristics, evaluate their quality, and incorporate them into the worldwide CT database maintained by the Cochrane Collaboration. Material and methods. CT were identified by a systematic, manual review of all the issues of MCILA. Descriptive analysis of their characteristics and methodological assessment of the CT found were performed. Results. In the period under consideration, 23 CT were published in MCILA. Most were lacking important information and the methodological quality was poor. Conclusion. To improve the quality and dissemination of CT published in Iberoamerican journals of dermatology, we recommend that authors and editors adhere to the international consensus initiatives for the publication of CT.

L10 ANSWER 26 OF 26 EMBASE

ACCESSION NUMBER: 2004453992 EMBASE
TITLE: Glucosamine improves symptoms of osteoarthritis, but further studies on its disease-modifying effects are needed.
SOURCE: Drugs and Therapy Perspectives, (2004) Vol. 20, No. 10, pp. 1-4. .
Refs: 13
ISSN: 1172-0360 CODEN: DTHPEE
COUNTRY: New Zealand
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Nov 2004
Last Updated on STN: 12 Nov 2004

AB Glucosamine occurs naturally in all human tissues. In the form of glucosamine sulfate, it has been studied in patients in osteoarthritis (OA). In 4-6 week clinical trials, oral and/or intramuscular glucosamine decreased OA symptom severity (as assessed by the Lequesne index) to a significantly greater extent than placebo and to a similar extent to ibuprofen. In 3-year placebo-controlled trials, glucosamine showed promising results in modifying the progression of OA. The tolerability profile of glucosamine sulfate is similar to that of placebo and better than that of ibuprofen or piroxicam. .COPYRGT. 2004 Adis Data Information

decubitus

Also found in: Medical

0.01 sec.

De`cu´bi`tus

Thesaurus

Legend: Synonyms | Related Words | Antonyms

Noun 1. decubitus - a reclining position (as in a bed)
posture, attitude, position - position or arrangement of the body and its limbs; "he assumed an attitude of surrender"

Mentioned in

bedsore
pressure sore
ulcer
ulceration

References in classic literature

No references found

Dictionary/thesaurus browser

Full browser

decriminalize
Decrown
Decrustation
decry
decrypt
decryption
Decubation
▸ **decubitus**
decubitus ulcer
Deculassement
Decuma
Decuman
Decumaria
Decumaria barbara
Decumaria barbata

DeCSS
DeCSS
DeCSS
DeCSS source code
DeCSS source code
DECstation
DECstation
DECSUPTO
DECsystem
DECsystem
Dect
Dect
Dect
DECT Multimedia Access Protocol
DECT-MMC
DECtalk
DECtape
DECtape
DECU
Decubation
decubital
Decubitis
Decubitis
Decubitis
▸ **decubitus**
decubitus calculus
decubitus paralysis
decubitus ulcer
decubitus ulcer
decubitus ulcer
decubitus ulcer
Decubitus ulcers
Decubitus ulcers
Decubitus ulcers
Decubitus ulcers
Decubitus ulcers
Deculassement
Decuma
Decuman
Decumanus
Decumanus Maximus
Decumaria
Decumaria
Decumaria barbara
Decumaria barbata
decumary
Decumbence
decumbency
decumbent
Decumbently

The Practical Peer-Reviewed Journal for Primary Care Physicians

Postgraduate Medicine online

symposium

The growing problem of pressure ulcers

Evaluation and management for an aging population

T. S. Dharmarajan, MD; Shamim Ahmed, MD

VOL 113 / NO 5 / MAY 2003 / POSTGRADUATE MEDICINE

CME learning objectives

- To understand the risk factors in the pathogenesis of pressure ulcers
- To understand the staging of pressure ulcers
- To learn principles of prevention and management of pressure ulcers, including general and local measures

The authors disclose no financial interests in this article.

This is the third of three articles on geriatric care.

Preview: Pressure ulcers, an important concern in older adults with restricted mobility, promise to become an even bigger issue as the US population ages. These ulcers can lead to devastating complications and place demands on an already stressed healthcare system. They also can be a quality indicator of the preventive measures taken in healthcare facilities. In this article, Drs Dharmarajan and Ahmed present guidelines for the prevention and treatment of pressure ulcers.
Dharmarajan TS, Ahmed S. The growing problem of pressure ulcers: evaluation and management for an aging population. Postgrad Med 2003;113(5):77-90

Persons who have cognitive impairment or restricted mobility, or both, are at risk for pressure ulcers. This potential consequence of functional impairment can result in increased use of healthcare resources (1). The prevalence of pressure ulcers in a particular facility is sometimes used as an indicator of the quality of the healthcare; inadequate use of preventive measures sometimes has been a basis for litigation (2,3). Prevention of these ulcers is key to their management. The US Department of Health and Human Services and the Agency for Health Care Policy and Research (AHCPR) have provided clinical practice guidelines for the prevention and treatment of pressure ulcers (4).

Definition and staging

A pressure ulcer is defined as any lesion caused by unrelieved pressure that damages underlying tissue (4). Because pressure is the primary pathophysiologic factor causing skin injury in typical ulcer locations, the term *pressure ulcer* is preferred to such synonyms as ***bedsore*** or *decubitus ulcer* (5). Typically, these ulcers occur over bony prominences. Because treatment differs, pressure ulcers must be distinguished from other conditions, such as **venous stasis** ulcers, ischemic lesions, and diabetic foot ulcers (2).

The staging system proposed by the National Pressure Ulcer Advisory Panel and the AHCPR is based on the extent of tissue damage (4). Skin injury from pressure ranges from blanchable erythema of intact skin to loss of full-thickness skin and damage to underlying tissue, including bone (table 1) (4,6). The four-stage system, which does not imply the progress of ulcers from one stage to another in the course of worsening or healing, has limitations (4). For example, stage I ulcers are not true ulcers because the skin is intact, and it can be difficult to delineate them in persons with dark skin. An ulcer cannot be accurately staged until its eschar is removed. Assessment of the skin may require removal of an orthopedic device (4).

Table 1. Characteristics of pressure ulcers used in staging

Stage I
Nonblanchable erythema of intact skin. Discoloration of skin, warmth, edema, or induration may be indicators in dark skin.

Stage II
Superficial ulcers involving loss of the epidermis, dermis, or both. These ulcers can present as an abrasion, blister, or shallow crater.

Stage III
Full-thickness skin loss involving damage to subcutaneous tissue that extends down to, but not through, underlying fascia

Stage IV
Full-thickness skin loss with extensive tissue destruction and necrosis of underlying muscle, bone, tendon, or joint capsule

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Adapted from Bergstrom et al (4).

Assessment should include documentation of anatomic location, stage, and description of size (ie, length, width, and depth). The presence of sinus tracts, undermining, maceration, exudate, or necrotic tissue and the presence or absence of granulation tissue should be noted (6). Pressure ulcers are most often found on the lower part of the body, particularly the bony prominences and weight-bearing surfaces of immobile persons (7). Areas prone to pressure ulcers include the sacrum, greater trochanter, ischial tuberosity, lateral malleolus, calcaneus, occiput, chin, ear, elbow, scapula, and iliac crest. Photographs of pressure ulcers may be considered part of assessment and documentation (8).

Prevalence

Pressure ulcers are common in older adults in the acute care setting, long-term care facilities, and community. Confinement to a bed or chair for a week increases the prevalence of these ulcers by 28% (5). Younger adults with spinal injuries also are vulnerable. When a stage I ulcer develops, the risk of additional ulcers increases tenfold (5). The prevalence of pressure ulcers is 3% to 11% in hospitalized adults, 2.5% to 24% in persons living in long-term care facilities, and up to 17% in adults in the community (5,8,9).

Pathophysiologic factors

When transient pressure interrupts blood flow, the skin becomes pale. If the ischemia lasts for more than a minute, the localized area becomes red or hyperemic, a reversible phenomenon, and blanchable. Nonblanchable erythema suggests capillary extravasation of plasma and red cells; it may be reversible if recognized promptly. Subcutaneous tissues, including muscles, are more sensitive to ischemia than the epidermis and dermis. Hence, pressure ulcers are more extensive than they appear superficially (6).

Risk factors

Identifying persons at high risk for pressure ulcers is vital (table 2). Unrelieved pressure is an essential risk factor that, coupled with inadequate microcirculation, results in tissue damage. Such ischemia is observed in persons of advanced age and in association with malnutrition, diabetes, cancer, terminal illness, sepsis, and vascular and neurologic disease (10). Risk factors are classified as either intrinsic or extrinsic (7,11).

Table 2. Risk factors for pressure ulcers

Intrinsic
Immobility
Limited functional ability
Fecal incontinence
Impaired sensation
Diminished level of consciousness
Poor nutritional status
Age, especially >75 yr
Comorbid conditions, including stroke, Parkinson's disease, fracture, sepsis, prior ulcers
Extrinsic
Pressure
Friction
Shearing
Moisture

Intrinsic

This type of risk factor relates to patient status. Immobility, limited functional ability, fecal incontinence, impaired sensation, and diminished level of consciousness place patients at higher risk for pressure ulcers (7,11). Geriatric patients who have fewer than 20 spontaneous nocturnal movements are at greatest risk; ulcers usually are not seen in patients who have more than 50 spontaneous nocturnal movements (5).

Hypoalbuminemia, a decreased lymphocyte count, decreased body weight, and inadequate dietary intake--all factors that suggest malnutrition--are associated with pressure ulcers. Age greater than 75 years appears to be a risk factor because changes in epidermal turnover, dermal thickness, collagen and elastin production, and vascularity occur during healthy aging (5,7). Stroke, contractures, Parkinson's disease, fractures, diabetes, sepsis, and prior ulcers add to the risk (5). Although fecal incontinence is considered a risk factor, the role of urinary incontinence remains controversial (12,13).

Extrinsic

Examples of this type of risk factor are pressure, friction, shearing, and moisture (7,11,14). Pressure, when unrelieved, is the most important factor in the pathogenesis of pressure ulcers. When a person lies on a standard hospital bed, the pressure over the greater trochanters and heels is 50 to 95 mm Hg; while the person is seated, pressure on the ischial tuberosity can be 300 to 500 mm Hg (5). These values are far above the normal capillary filling pressure of 32 mm Hg, and such pressure occludes circulation (11,14). Pressure of 60 to 70 mm Hg for 1 or 2

hours may lead to muscle injury (5,11).

In a healthcare facility, friction can occur when a patient is pulled across support surfaces during transferring and repositioning, causing loss of stratum corneum and the formation of intraepidermal blisters. When the blisters are unroofed, superficial erosions, skin tears, or abrasions (ie, stage II ulcers) become apparent (5,11,14).

Shearing forces are exerted when the skin and subcutaneous tissues slide on each other when the patient's head is elevated more than 30° or when gravity causes a seated patient to slide down. Sliding causes angulation of perforating arterioles, which compromises circulation (5,11,14). Moisture softens the stratum corneum and increases the risk of maceration injury with friction. Acid and bacterial products from urine or feces may contribute to the problem (7).

Impact on morbidity

Pain is a severe or distressing symptom in half of persons who have pressure ulcers, but only a small number receive adequate analgesia (5). Local infection that manifests as cellulitis or osteomyelitis is the most common infectious complication. Osteomyelitis occurs in up to one fourth of nonhealing ulcers (5). Sepsis is associated with high mortality (5). Other complications include bacteremia, endocarditis, meningitis, septic arthritis, sinus tract or abscess, amyloidosis, and squamous cell carcinoma in the ulcer (4).

Prevention

The AHCPR recommendations to prevent pressure ulcers, which are listed in table 3, include risk assessment, measures to relieve pressure, proper skin care and nutrition, and steps to minimize moisture from urinary and fecal incontinence (4).

Table 3. Prevention of pressure ulcers

Risk assessment
Identify person predisposed to pressure ulcers

Measures to relieve pressure
Turn or reposition often
Use positioning devices
Use support surfaces

- Static: mattresses or overlays (foam, gel, water, air)
- Dynamic: low-air-loss and air-fluidized beds

Nutritional assessment and supplements

Skin care
Cleanse with isotonic sodium chloride solution
Avoid rubbing or massaging ulcers
Prevent excessive dryness by avoiding hot water and soap
Protect from excessive moisture

Management of urinary and fecal incontinence

Adapted from the American Medical Directors Association (19).

Risk assessment
Risk assessment should be performed to identify patients who are bound to a bed or wheelchair and therefore are predisposed to pressure ulcers. The AHCPR guidelines recommend the use of systematic risk assessment tools, such as the Braden scale or Norton scale, and reassessment at regular intervals (4). The Braden scale evaluates the level of sensory perception, skin moisture, physical activity, mobility, food intake, friction, and shear. The Norton scale assesses physical condition, mental state, activity, mobility, and incontinence (8,11,15).

Measures to relieve pressure
Turning and positioning, use of support surfaces, and proper skin care and nutrition can minimize the occurrence of pressure ulcers.

Turning and positioning: Frequent repositioning along with the use of support surfaces helps limit the amount and time of pressure exposure. Observational studies have shown that patients should be turned every 2 hours to limit ischemia to tissues overlying bony prominences. Even seated patients need to be repositioned often to relieve pressure. Individualized and specialized seat cushions are beneficial.

Positioning devices help maintain a position and prevent contact of bony prominences with one another. Because the heel is susceptible, total pressure relief with pillows placed under the calf is recommended. During repositioning, a patient may be placed at 30° angles (oblique position) to reduce pressure on the trochanter. To minimize shearing, the lowest degree of head elevation is recommended. The use of lifting devices reduces friction-associated injury (4,14,15).

Use of support surfaces: The use of a pressure- reducing surface lowers the incidence and severity of pressure ulcers (4,14). Support surfaces are static or dynamic depending on their ability to alternate the tissue interface pressure independent of the patient. Pads, cushions, and most mattress overlays are static; some mattress overlays, special mattresses, and specialty beds (low-air-loss and air-fluidized beds) are dynamic (14). Sheepskin, egg-crate foam, and 2-in foam pads are inexpensive. However, they are unable to reduce pressure sufficiently (5); high-density, 4-in foam overlays are more effective at lowering interface pressure (5).

Static air mattresses or overlays feature interconnected air cells that deflate or inflate with patient movements and equalize interface pressure. Dynamic air-filled products have a series of air cells that are alternately inflated and deflated

with a bedside pump, intermittently relieving pressure at bony sites (5,14). These alternating-pressure devices decrease skin pressure significantly. Water mattresses are useful to reduce pressure, but they may increase the risk of maceration because they are made of impermeable materials. These mattresses are not often used because they are heavy and may leak (5,14).

A specialized bed should be considered for patients who have multiple large, stage III or IV ulcers to prevent new ulcers and accelerate healing of existing ones (5). Low-air-loss beds consist of air-permeable fabric pillows that are constantly inflated with air, have a drying effect on tissues from a flow of warm air, and allow several different positionings. Air-fluidized beds contain microspheric silicon beads covered by an air-permeable fabric; streams of warm air forced through the beads allow the patient to float on the beads. Considerable variations in dermal-insensible fluid loss that can affect body temperature and hydration necessitate monitoring of these parameters (5,14,16).

Skin care

Systematic skin inspection should be performed at least once a day, and the skin should be cleansed at regular intervals and whenever it is soiled. Hot water and drying soaps are best avoided. To treat dry, flaky, scaly skin, optimum environmental humidity should be maintained and moisturizers used. Areas of redness over bony prominences should not be massaged or rubbed. Mobility through rehabilitation should be encouraged (15).

Nutrition

Nutritional assessment is an important part of evaluation. Pressure ulcers and malnutrition often coexist, but a causal relationship is not well established. A serum albumin level of less than 3.5 g/dL, a total lymphocyte count less than 1,800/microliter, and an unintentional reduction in body weight suggest malnutrition (4).

The goal is to provide about 30 to 35 calories and 1.25 to 1.50 g of protein per kilogram per day (4). A daily high-potency multivitamin and mineral supplement is recommended when micronutrient deficiency is suspected (4). Short-term supplementation with vitamin C (500 mg/day) and zinc (15 mg/day) may promote ulcer healing in the presence of deficiency (2,4).

Although malnutrition is potentially reversible, nutritional support by itself does not invariably lead to the healing of ulcers. The decision to administer parenteral or enteral supplements should be made on the basis of the patient's overall status, life expectancy, and quality of life and the preferences of the patient or caregiver (5,15).

Moisture

Moisture from urine, stool, and perspiration can lead to skin damage (7,15). Bacterial colonization is common in the setting of fecal incontinence. Urinary incontinence should be addressed. Moisture barriers (eg, liquid spray barriers, petroleum jelly, transparent adhesive dressings) are helpful after skin cleansing (15,17).

Treatment

Proper documentation of wound characteristics (see table 1) is essential, along with history taking, physical examination, and assessment of complications (4,18). Comorbid illnesses, such as diabetes, peripheral vascular disease, immune deficiency, collagen vascular disease, malignancy, psychosis, and depression, deserve evaluation (4).

A treatment plan should consist of the previously described preventive measures as well as specific ulcer care, infection control, and monitoring at regular intervals (table 4) (4,18). Patients with cognitive impairment may not complain of pain, but when pain is suspected, analgesic relief is important to improve their quality of life (4,18). Other crucial measures include appropriate wound covering, adjustment of support surfaces, and repositioning.

Table 4. Treatment of pressure ulcers

General

Ulcer staging and documentation

Provision of adequate nutrition

Assessment for complications

Pain management

Tissue load reduction

Management of local and systemic infection

Surgical repair

Local

Stage I

Prevention of further skin damage

Stage II

Debridement usually not necessary

Cleansing with isotonic sodium chloride solution and avoidance of high-pressure irrigation

Use of hydrocolloid wafers, semipermeable foam dressing, or polyurethane film

Stages III and IV

Debridement with autolytic, mechanical (wet-to-dry dressing), enzymatic, or surgical methods

Cleansing with isotonic sodium chloride solution, using high-pressure irrigation if necessary

Dressing: If wound is shallow and clean, use hydrocolloid

wafers, semipermeable foam dressing, or polyurethane film. If wound is deep and clean, fill dead space with wet gauze. If there is necrotic debris, use wet-to-dry dressing. If there is excessive exudate, use absorptive dressing.

Local wound care

Local care involves debridement, wound cleansing, and application of dressings. Some cases need adjunct therapy and surgical repair (2,4,18,19). No specific treatment is required for stage I ulcers in which the skin is intact. The goal of treating these ulcers is to preserve intact skin and prevent deep-tissue damage (2,18).

Because stage II ulcers are shallow, debridement usually is unnecessary. If minimal necrotic tissue is present, autolytic debridement under occlusive or semipermeable dressings is adequate. Wounds should be cleansed with isotonic sodium chloride solution, but high-pressure irrigation should be avoided to minimize injury to healthy tissue. Optimal dressings include hydrocolloid or hydrogel wafers, semipermeable foam dressing, and polyurethane film that is changed every 3 to 7 days. Occlusive dressings help epithelial migration and keep surrounding skin dry while maintaining a moist wound bed (2,18).

Stage III and IV ulcers usually require debridement because they contain devitalized tissue and necrotic debris. In the setting of cellulitis, bacteremia, or sepsis, prompt surgical debridement is essential. Methods of debridement are autolytic, mechanical, enzymatic, and sharp (surgical) (18,19).

Autolytic debridement: In this process, the ulcer is covered with a transparent adhesive, hydrocolloid wafer, or semipermeable foam wafer that allows accumulation of tissue fluids that contain macrophages, neutrophils, and enzymes, which selectively remove bacteria and devitalized tissue (18,19).

Mechanical debridement: This type can be accomplished by the use of classic wet-to-dry dressings. A moist dressing is applied and allowed to dry and adhere to tissue at the wound base. As the dressing is removed, it also removes the adherent tissue. Topical enzymes (eg, collagenase [Collagenase Santyl], fibrinolysin) are available to remove devitalized tissue. Wounds with necrotic debris are cleansed with high-pressure irrigation using a 35-mL syringe and a 19-gauge angiocatheter (18). If an ulcer is shallow and clean, hydrocolloid wafers or polyurethane film may be the choice of dressing after cleansing; when the ulcer is deep, the dead space should be filled with wet gauze and kept continuously moist. Alginate dressings are highly absorbent and useful in exudating ulcers (18).

Sharp debridement: This type, which is also referred to as surgical debridement, involves the use of sterile surgical instruments to remove nonviable tissue and is indicated in the presence of advancing cellulitis, thick eschar, or extensive necrosis (20). Healthcare professionals who perform sharp debridement must have the necessary skills and meet state licensing requirements. Sharp debridement

is the fastest form of debridement and can be performed at the bedside or in the operating room with sterile forceps, scalpel, or scissors.

Most types of debridement cause significant pain that requires adequate analgesia during and after the procedure. Because bacteremia can occur during debridement, the need for prophylactic antibiotics, particularly for endocarditis, should be considered (4,19,20).

Infection control

Stage II, III, and IV ulcers are invariably colonized with bacteria (4). Effective wound cleansing and debridement minimize colonization and enhance healing. Swab cultures are not suggested, because all ulcers are colonized by bacteria (4). For nonhealing ulcers, a 2-week trial of topical antibiotics (eg, silver sulfadiazine [Silvadene, SSD, Thermazene], triple-antibiotic ointment) may be tried (4,21). When advancing cellulitis or osteomyelitis is suspected, soft-tissue culture by tissue biopsy or needle aspiration may be performed.

Gram-negative bacilli or anaerobic bacteria may cause sepsis from nonhealing infected ulcers; older adults, especially long-term care facility residents with pressure ulcers, are vulnerable to tetanus infection (20-22). Topical antiseptics (eg, povidone iodine, iodophor, sodium hypochlorite solution, hydrogen peroxide) are discouraged because they are toxic to healthy granulation tissue (4). Systemic antibiotics are indicated for bacteremia, sepsis, cellulitis, and osteomyelitis (4).

Surgical repair

When clean stage III or IV pressure ulcers in medically stable patients do not respond to optimal care, surgical repair may be considered. Methods include direct closure, skin grafts, skin flaps, musculocutaneous flaps, and free flaps (4,19).

Adjuvant therapy

Adjuvant therapy includes electrical stimulation, exposure to hyperbaric oxygen or infrared or ultraviolet light, low-energy laser irradiation, ultrasound therapy, and application of topical agents (eg, growth factors, maggot therapy) (4,19,23). Maggot infestation is a potential complication of pressure ulcers. However, sterile maggots are sometimes used for debridement and may be successful, although patients find them uncomfortable and aesthetically displeasing. Only electrical stimulation therapy has been recommended by the AHCPR as an adjunct to conventional therapy for nonhealing ulcers (4).

Conclusion

Pressure ulcers are a common and frustrating problem in the geriatric population. They increase demands on healthcare resources and are sometimes a source of malpractice litigation. The timely institution of prophylactic and corrective measures can help prevent or slow the progression of pressure ulcers. When ulcers do occur, proper selection from many available therapies can prevent potentially devastating complications.

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INTRODUCTION

The terms **decubitus** ulcer and pressure sore often are used interchangeably in the medical community. **Decubitus**, from the Latin *decumbere,* means "to lie down." **Decubitus** ulcer, therefore, does not adequately describe ulceration that occurs in other positions, such as prolonged sitting (eg, the commonly encountered ischial tuberosity ulcer). Because the common denominator of all such ulcerations is pressure, pressure sore is the better term to describe this condition.

History of the Procedure: Pressure sores have probably existed since the dawn of our infirm species. They have been noted in unearthed Egyptian mummies and addressed in scientific writings since the early 1800s. Presently, treatment of pressure sores in the United States is estimated to cost in excess of $1 billion annually.

Problem: Pressure is exerted on the skin, soft tissue, muscle, and bone by the weight of an individual against a surface beneath. These pressures are often in excess of capillary filling pressure, approximately 32 mm Hg. In patients with normal sensitivity, mobility, and mental faculty, pressure sores do not occur. Feedback, both conscious and unconscious, from the areas of compression leads individuals to change body position. These changes shift the pressure prior to any irreversible tissue damage.

Individuals unable to avoid long periods of uninterrupted pressure over bony prominences are at increased risk for the development of necrosis and ulceration. This group of patients typically includes elderly individuals, those who are neurologically impaired, and those who are acutely hospitalized. These individuals cannot protect themselves from the pressure exerted on their bodies unless they consciously change position or have assistance in doing so. Even the most conscientious patient with an extensive support group and unlimited financial resources may develop ulceration resulting from a brief lapse in avoidance of the ill effects of pressure.

Frequency: Two thirds of pressure sores occur in patients older than 70 years. The prevalence rate in nursing homes is estimated to be 17-28%.

Among patients who are neurologically impaired, pressure sores occur with an annual incidence of 5-8%, with lifetime risk estimated to be 25-85%. Moreover, pressure sores are listed as the direct cause of death in 7-8% of all paraplegics.

Patients hospitalized with acute illness have an incidence rate of pressure sores of 3-11%.

Disturbingly, even with current medical and surgical therapies, patients who achieve a healed wound have recurrence rates of as high as 90%.

Etiology: Many factors contribute to the development of pressure sores, but

pressure leading to ischemia is the final common pathway. Tissues are capable of withstanding enormous pressures when brief in duration, but prolonged exposure to pressures slightly above capillary filling pressure initiates a downward spiral towards ulceration.

Impaired mobility is an important contributing factor. Patients who are neurologically impaired, heavily sedated, restrained, or demented are incapable of assuming the responsibility of altering their position to relieve pressure. Moreover, this paralysis leads to muscle and soft tissue atrophy, decreasing the bulk over which these bony prominences are supported.

Contractures and spasticity often contribute by repeatedly exposing tissues to pressure through flexion of a joint. Contractures rigidly hold a joint in flexion, while spasticity subjects tissues to considerable repeated friction and shear forces.

Sensory loss also contributes to ulceration by removing one of the most important warning signals, pain.

Paralysis and insensibility also lead to atrophy of the skin with thinning of this protective barrier. The skin becomes more susceptible to minor traumatic forces, such as friction and shear forces, exerted during the moving of a patient. Trauma causing deepithelialization leads to transdermal water loss, creating maceration and adherence of the skin to clothing and bedding, which raises the coefficient of friction for further insult.

Malnutrition, hypoproteinemia, and anemia reflect the overall status of the patient and can contribute to vulnerability of tissue and delays in wound healing. Poor nutritional status certainly contributes to the chronicity often observed with these lesions. Anemia indicates poor oxygen-carrying capacity of the blood. Vascular disease also may impair blood flow to the region of ulceration.

Bacterial contamination from improper skin care or urinary or fecal incontinence, while not truly an etiological factor, is an important factor to consider in the treatment of pressure sores and can delay wound healing.

Pathophysiology: The inciting event is compression of the tissues by an external force such as a mattress, wheelchair pad, or bed rail. Other traumatic forces that may be present include shear forces and friction. These forces cause microcirculatory occlusion as pressures rise above capillary filling pressure, resulting in ischemia. Ischemia leads to inflammation and tissue anoxia. Tissue anoxia leads to cell death, necrosis, and ulceration.

Irreversible changes may occur after as little as 2 hours of uninterrupted pressure.

Clinical: Clinical presentation of pressure sores can be quite deceiving to the inexperienced observer. Soft tissues, muscle, and skin have a differential resistance to the effects of pressure. Generally, muscle is the least resistant and will necrose prior to skin breakdown. Also, pressure is not equally distributed from the bony surface to the overlying skin. Pressure is greatest at the bony prominence, decreasing gradually towards the periphery. Once a small area of skin breakdown has occurred, one may be viewing only the tip of the iceberg, with a large cavity and extensive undermining of the skin edges.

Many classification systems for staging pressure ulcers have been presented in the literature. The most widely accepted system is that of Shea, which has been modified to represent the present National Pressure Ulcer Advisory Panel classification system. This system consists of 4 stages of ulceration but is not intended to imply that all pressure sores follow a standard progression from stage I to stage IV. Nor does it imply that healing pressure sores follow a standard regression from stage IV, to stage I, to healed wound. Rather, it is a system designed to describe the depth of a pressure sore at the specific time of examination, to facilitate communication among the various disciplines involved in the study and care of such patients.

Stage I represents intact skin with signs of impending ulceration. Initially this would consist of blanchable erythema from reactive hyperemia that should resolve within 24 hours of the relief of pressure. Warmth and induration also may be present. Continued pressure creates erythema that does not blanch with pressure. This may be the first outward sign of tissue destruction. Finally, the skin may appear white from ischemia.

Stage II represents a partial-thickness loss of skin involving epidermis and possibly dermis. This lesion may present as an abrasion, blister, or superficial ulceration.

Stage III represents a full-thickness loss of skin with extension into subcutaneous tissue but not through the underlying fascia. This lesion presents as a crater with or without undermining of adjacent tissue.

Stage IV represents full-thickness loss of skin and subcutaneous tissue and extension into muscle, bone, tendon, or joint capsule. Osteomyelitis with bone destruction, dislocations, or pathologic fractures may be present. Sinus tracts and severe undermining commonly are present.

Other important characteristics of the wound should be noted in addition to depth. One should note the presence or absence of foul odors, wound drainage, eschar, necrotic material, and soilage from urinary or fecal incontinence. This provides information regarding the level of bacterial contamination and the need for debridement or diversionary procedures.

The overall state of health, comorbidities, nutritional status, mental status, and smoking history also should be noted. Presence or absence of contractures and spasticity also are important in the formulation of a treatment plan. One should note where the patient normally resides and the extent of his or her support structure. Examining the support surfaces present on the patient's bed or wheelchair also is important.

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Relevant Anatomy: A frequency among anatomic sites exists in affected individuals. The hip and buttock regions account for 67% of all pressure sores, with ischial tuberosity, trochanteric, and sacral locations being most common. The lower extremities account for an additional 25% of all pressure sores, with malleolar, heel, patellar, and pretibial locations being most common.

The remaining 10% or so of pressure sores may occur in any location that experiences long periods of uninterrupted pressure. Nose, chin, forehead, occiput, chest, back, and elbow are among the more common of the infrequent sites for pressure ulceration. No surface of the body can be considered immune to the effects of pressure.

Contraindications: See Ethical considerations.

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Diagnostic Procedures:

- Differentiation of bacterial infection from simple contamination is best made with a tissue biopsy, which allows quantitative wound culture techniques. This will indicate whether antibiotics should be administered.

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Medical therapy: The first step in resolution is to reduce or eliminate the cause, ie, pressure. Specialized support surfaces are available for bedding and wheelchairs, which can maintain tissues at pressures below 30 mm Hg. These specialized surfaces include foam devices, air-filled devices, low-airloss beds (Flexicair, KinAir), and air-fluidized beds (Clinitron, FluidAir). Low-airloss beds support the patient on multiple inflatable air-permeable pillows. Air-fluidized beds suspend the patient as air is pumped into an air-permeable mattress containing millions of microspheric uniformly sized silicone-coated beads. No one device has been shown to be clearly superior over the others, but they all have been shown to reduce tissue pressure over conventional hospital mattresses and wheelchair cushions. Over 75 companies sell pressure-reduction devices, with annual industry revenues in excess of $8 billion.

Regardless of the choice of support surface, turning and repositioning the patient remain the cornerstones of prevention and treatment. This should be performed every 2 hours, even in the presence of a specialty surface or bed.

The wound and surrounding skin must be kept clean and free of urine and feces. This should be done through frequent cleansing and the establishment of a bowel and bladder regimen. Constipating agents may be helpful. Bacterial contamination must be assessed and treated appropriately. Differentiation of bacterial infection from simple contamination is best made with a tissue biopsy, which allows quantitative wound culture techniques. This will indicate whether antibiotics should be administered.

Wound dressings vary with the state of the wound. A stage I lesion with signs of impending breakdown may require no dressing. Stage II ulcers confined to the epidermis or dermis may be treated with a hydrocolloid occlusive dressing (DuoDerm), which maintains a moist environment to facilitate reepithelialization. For more advanced ulcers, a large variety of treatment options is available. These include wet-to-dry dressings, incorporating isotonic sodium chloride solution or dilute Dakins solution (sodium hypochlorite), Silvadene, Sulfamylon, hydrogels (Carrington gel), xerogels (Sorbsan), and vacuum-assisted closure (VAC) sponges. Daily whirlpool use also may serve to irrigate and mechanically debride the wound.

The choice of treatment and dressings is not as important as their appropriate application. These dressings are not a substitute for sharp debridement in severely contaminated wounds with necrotic material. Although uncommon, grossly infected pressure sores can lead to sepsis, myonecrosis, necrotizing fasciitis, and gangrene if not adequately debrided.

Spasticity should be relieved with diazepam, baclofen, dantrolene sodium, mephenesin carbonate, dimethothiazine, or orciprenaline. Flexion contractures may be relieved surgically.

Nutritional status should be evaluated and optimized. This is one of the only contributing factors that may be considered reversible. This may require dietary supplements, enteral feedings, or even parenteral feedings. Restoring a positive nitrogen balance and a serum protein level of 6 mg per 100 mL or higher has been shown to facilitate wound healing.

A multidisciplinary approach can lead to maximum benefit for the patient. Consultations with a neurosurgeon, urologist, plastic surgeon, orthopedic surgeon, and general surgeon all may be indicated in a particular patient. A rehabilitation medicine specialist, social worker, and psychologist or psychiatrist may work together with geriatricians or internists to improve the patient's health, attitude, support structure, and living environment.

When medical management has been optimized, many stage I and stage II pressure sores heal spontaneously. However, stage III and stage IV ulcers almost always require a surgical approach. Plastic surgeons perform most pressure sore reconstructions, and consulting a plastic surgeon with any complex or chronic wound is appropriate.

Surgical therapy: Even with optimal medical management, many patients require a trip to the operating room for debridement, diversion of urinary or fecal stream, release of flexion contractures, wound closure, or amputation.

Debridement is aimed at removing all devitalized tissue that serves as a reservoir for ongoing bacterial contamination and possible infection. Extensive debridement should be done in the operating room, but minor debridement is commonly performed at the bedside. Although many of these patients are insensate, others are unable to communicate pain sensation due to underlying disease processes. Pain medication should be administered liberally, and vital signs often are a good indicator of pain perception. Care also should be taken when debriding at the bedside because wounds may bleed significantly.

Urinary or fecal diversion may be necessary to optimize wound healing. Many of these patients are incontinent and their wounds are contaminated with urine and feces daily. Patients with loose stools benefit from constipating agents and a low-residue diet.

Release of flexion contractures resulting from spasticity may assist with positioning problems, and amputation may be necessary for a nonhealing wound in a patient who is not a candidate for reconstructive surgery.

Reconstruction of a pressure ulcer is aimed at improvement of patient hygiene and appearance, prevention or resolution of osteomyelitis and sepsis, reduction of fluid and protein loss through the wound, and prevention of future malignancy (Marjolin ulcer).

Preoperative details: The concept that medical management must be optimized prior to surgical reconstruction of a pressure sore cannot be overemphasized; otherwise, reconstruction is doomed to failure. This means that spasticity must be controlled, nutritional status must be optimized, and the wound must be clean and free of infection.

Two units of type-specific packed red blood cells should be available during the operation because blood loss may be significant.

Intraoperative details: Patient positioning is dictated by the location of the ulcer and the planned reconstruction. Many pressure sores occur in the gluteal region and require prone positioning. Most anesthesiologists choose to use general endotracheal anesthesia, particularly if the patient is prone, but ulcer closure may be performed under regional or local anesthesia if necessary.

The first step is to adequately excise the ulcer. This includes the bursa or lining of the ulcer, surrounding scar, and any heterotopic calcification found. Underlying bone must be adequately debrided to avoid a retained nidus of osteomyelitis. Some evidence in the literature indicates that pulsed lavage can be beneficial in reducing bacterial counts in wounds, and some surgeons routinely employ this method following debridement.

Once appropriately debrided, the wound may be closed in a variety of ways depending on the location of the pressure sore, previous scars or surgeries, and surgeon preference. However, the tenets of reconstruction remain the same in all pressure sore reconstructions.

Very few pressure sores can or should be closed primarily following debridement due to unacceptably high complication rates. A well-vascularized pad of tissue should be placed in the wound. This tissue usually is a musculocutaneous flap transposed or rotated on a pedicle containing its own blood supply. This also may involve the use of tissue expansion or a free flap with microvascular anastomosis. The purpose of this tissue is to eliminate dead space within the wound, enhance perfusion, decrease tension on the wound closure, and provide a new source of padding over the bony prominence.

Prior to wound closure, drains should be placed in the bed of the wound. This allows external drainage of any fluid that may accumulate beneath the flap and hopefully avoids wound complications such as hematoma or seroma.

Postoperative details: The ultimate success or failure of pressure sore reconstruction only begins in the operating room. Wound healing and prevention of recurrence become the goals following successful closure of a pressure sore.

Postoperatively, the patient should be maintained on a specialized support surface for no fewer than 6 weeks. This may be in the hospital, at a rehabilitation facility, or at home.

After approximately 6 weeks, at the discretion of the surgeon, patients may gradually reintroduce temporary pressure to the surgical site by sitting. The patient must accept the responsibility that he or she never again sits for more than 2 hours in one position.

Perform skin care daily. This involves a careful inspection of all skin surfaces to identify areas of impending breakdown prior to their occurrence. Skin should be washed with soap and water and completely dried. Moisture should not be allowed to accumulate on the skin or in clothing or bedding, nor should the skin be allowed to become overly dry and scaly. Skin moisturizers are useful to maintain the appropriate level of moisture at the surface of the skin.

Control of spasticity and maintenance of adequate nutrition also must be continued into the outpatient setting to prevent recurrence.

Follow-up care: Follow-up should be performed every 3 weeks for the first several months. The interval may then be increased to every 6 months and then yearly. Early issues include suture removal, drain removal, and when to allow the patient to exercise or sit up.

Once healing is complete, long periods of uninterrupted pressure must be avoided. This involves frequent repositioning by the patient or their support group. Seated patients with upper extremity function should lift themselves from their wheelchair for at least 10 seconds every 10-15 minutes. Patients in bed should be repositioned at least every 2 hours.

Pressure dispersion, through the application of specialized support surfaces on beds and wheelchairs, should be extended through the wound healing period and into the outpatient setting if available and tolerated by the patient. This is an adjunct to the alternating of weight-bearing surfaces and maintains low pressures on the tissues at all times.

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Complications fall into 1 of 2 categories: complications of chronic ulceration and complications of ulcer reconstruction.

The most serious complication of chronic ulceration is malignant degeneration, or Marjolin ulceration. Initially described by Marjolin in 1828 as a cancer arising in burn scars, malignant degeneration has been reported in patients with chronic pressure sores. These malignancies typically are highly aggressive squamous cell carcinomas with a high likelihood of nodal metastasis at the time of diagnosis. Any long-standing nonhealing wound should alert the examiner to the need for biopsy.

Complications as a result of reconstructive surgery are, unfortunately, considerable. These include hematoma, seroma, wound dehiscence, wound infection, and recurrence. Due to the use of well-vascularized flaps, flap necrosis is infrequent.

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Achieving a closed wound is the beginning of a lifelong struggle to prevent recurrent ulceration at the surgery site or at a new site. Recurrence rates in the literature are reported to be as high as 90%.

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Promising research in the field of growth factors and wound healing has shed light on the complex interactions that ensue at the wound surface and in the affected organism as a whole. This has led to the introduction of becaplermin (Regranex), recombinant human platelet-derived growth factor. This topical agent has been approved by the US Food and Drug Administration (FDA) for the treatment of lower extremity diabetic neuropathic ulcers that extend into the subcutaneous tissue or beyond. Studies are underway to possibly expand the approved indications for this drug to include other wounds. Other growth factors also are being evaluated for use in human clinical settings. This expanding field surely will contribute further applications of basic science to clinical wound healing, with improvement of our understanding and patient care.

Ethical considerations

As a final note, one should consider the ethics of pressure sore treatment. The aggressive treatment of

pressure ulceration is outlined in this article. This treatment certainly is indicated for one subset of patients who have pressure ulceration, ie, the acutely hospitalized patient with a recoverable illness.

For others, such as chronically or terminally ill patients with long-standing or recurrent ulceration, aggressive treatment may not be in the best interest of the patient. In these instances, the wishes of the patient or the patient's family should be weighed carefully. In many instances, medical care and maintaining patient comfort should be the goals rather than the institution of major invasive procedures.

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